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RETROBULBAR NEURITIS AND DISEASE OF THE NASAL ACCESSORY SINUSES

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The function of the optic nerves and optic tracts is to convey the various impulses of light stimuli from the retinas to the cortex of the brain and to carry them uninterruptedly with regard to strength, quality and duration of the stimulus. The structure of the optic nerve is such that interruption of stimuli in isolated bundles of fibers may occur with little if any interference with the proper function of the remainder of the nerve, a condition that is projected in the visual field as scotoma or anopsia. Knowledge of the position of various bundles of fibers in the nerve, with reference to the retinal elements and their projection into the visual field, is used as a basis for determining the situation of obstructions along the visual pathways. Of particular significance are homonymous anopsia and scotoma. When only one eye is involved, or when one eye is blind, the localizing value of the field chart is somewhat less. In addition to the field defects, which are studied by means of the perimeter and campimeter, further information concerning the situation of a lesion is obtained through ophthalmoscopic examination of the fundus, which reveals the presence or absence of pathologic change in the nerve head. Central visual acuity, a function chiefly dependent on the condition of the retina, nerve and tracts, is an important item in the data required for understanding the nature of lesions of the optic pathways, and in certain cases becomes of considerable importance in differential diagnosis.

Disturbance of vision constitutes the chief symptom of conditions which affect the optic nerves, tracts and radiations, and the cause of such disturbance may lie entirely outside the nerve. Anatomic anomalies, such as abnormally placed bony structures, abnormal blood vessels, oxycephaly and dolichocephaly, may, in the course of development incident to growth, cause pressure on the nerve or stretch it in such a way as to produce partial or complete atrophy.

Interruption of function of a portion of the optic nerve, producing central or peripheral scotoma, or anopsia, results from direct pressure

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on the nerve by tumors of the nerve or its sheath and by cysts or tumors adjacent to the chiasm, a well known example of which is tumor of the pituitary gland. Tumors of the brain, situated more remotely, may press the chiasm downward against the bony sella or the large vessels, producing indentations in the chiasm resulting in partial blockage. This pressure interferes with the blood supply of the nerve, as is seen in pallor of the optic disk without loss of nerve substance, a characteristic ophthalmoscopic finding in tumor of the pituitary gland. If the pressure is severe for a relatively short time, or if mild pressure persists long enough, atrophy of the nerve will occur, with permanent loss of vision. But if the pressure is removed, or if the blood supply to the nerve is sufficiently increased without removing the pressure, the function of the nerve will be restored to that portion which has not been permanently injured and has not already become atrophic. It is important to understand that the optic nerve will recover from the effects of injury sufficient to cause temporary total blindness, whether from outside sources such as tumor of the brain, or from swelling and infiltration of the septal network of tissue within the nerve trunk, which occurs, for example, in multiple sclerosis. The axis-cylinders will withstand direct pressure or ischemia for a considerable time before final degeneration begins, and will gradually resume their interrupted function when the causes of their temporary blocking are removed. This phenomenon constitutes the basis for all forms of treatment of optic neuritis, and is of primary significance in a consideration of treatment of retrobulbar neuritis from whatever cause.

Disease of the nerve itself accounts for the largest number of cases of retrobulbar neuritis if it is assumed that multiple sclerosis is a primary disease of the nervous system. Other primary degenerative lesions may also be considered, although they occur less commonly and are often part of a more extensive disorder, such as Schilder's disease.

The toxic effect on vision and the ultimate change in the nerve that may be caused by such substances as alcohol, nicotine, lead, quinine, arsenic and thallium are well known. Much has been said of the effect of endogenous toxins associated with acute contagious diseases, influenza, tuberculosis, syphilis, intestinal toxemia and particularly disease of the paranasal sinuses, the toxin of which is reported to be effective through contiguous tissue or by transmission through the blood stream or lymphatic structures. I shall refer to this again.

The typical syndrome of retrobulbar neuritis is not often missed. It consists of lowered visual acuity, some form of scotoma in the visual field, usually central scotoma, and a nerve head of normal appearance. The inaccuracy of the designation, "retrobulbar neuritis," leads to confusion in trying to visualize the lesion along the pathway, and connotes its situation somewhere between the globe and the optic chiasm, when, as a matter of fact, the lesion that causes anopsias often lies in the chiasm or posterior to it. The realization of that defection in nomenclature does

not prevent a widespread conviction that operations on the sinuses should be undertaken early. The reported results of such operations, when taken at their intended value, vitiate conclusions based on unprejudiced observation of this and all other methods of treatment. Arguments based on findings which are improperly interpreted are formulated to bolster a therapeutic procedure inconsistent with findings in a similar group of patients treated in a different manner. Operations on healthy paranasal sinuses for retrobulbar neuritis are still advocated by some ophthalmologists. In many cases the results have been good, but the reasons for the good results may be attributed to other factors than the one usually given, namely, drainage of secretions from the sinuses.

It is essential to obtain a careful history of the onset and course of visual disturbances. Loss of vision may be sudden or gradual. In cases caused by inflammation it usually is acute. Within a few hours, often overnight, the vision of one eye may be completely obliterated. In many cases, perception of moving objects returns within a day or two, and gradually the peripheral field improves, with persistence of a defect in the central field. In mild cases, the loss of vision may extend slowly for several days, varying in extent and amount, but with predominance in the central or centrocecal area. The simultaneous occurrence of symptoms in both eyes is rare, although one eye may be severely affected, whereas the visual acuity of the other may be only slightly diminished.

Recovery in acute cases usually is rapid, beginning a few days after the onset; improvement continues for from four to eight days, when progress seems to be arrested. The central vision remains dim, or even entirely absent, whereas peripheral vision for form and color are well restored to within 10 to 20 degrees of the center. In many cases of acute onset and in cases of mild intensity, central vision may remain almost stationary for days or even weeks. In others, the loss of vision is so gradual that, when first noticed, the defect in the peripheral field is well advanced, particularly in cases of anopsia. Total permanent blindness may be reached only after weeks or months of slowly progressive contraction of the field, or expansion of the central scotoma. Complete recovery may be followed by another episode of amblyopia, of greater or lesser severity, at intervals of as long as ten years, as was seen in two cases of multiple sclerosis at the Mayo Clinic. Adie [1] reported a recurrence after twenty-four years, and wrote that "the longest reported case is forty-seven years." Spontaneous recovery of vision is often seen in retrobulbar neuritis caused by multiple sclerosis and alcohol and nicotine poisoning, and in other cases of undetermined origin.

Pain is not a common symptom in retrobulbar neuritis. Tenderness of the globes, elicited by rotation of the eyes or by digital pressure, is a minor symptom elicited by questioning, but seldom volunteered by the

1. Adie, W. J.: Acute Retrobulbar Neuritis in Disseminated Sclerosis, Tr. Ophth. Soc. U. Kingdom **50**:262, 1930.

patient. Soreness of the neck, and slight pain in the back of the head are mentioned occasionally, but are not to be considered the usual symptoms of optic neuritis of any type. Friedenwald [2] has noted that a good number of cases of optic neuritis is ushered in with slight fever, general malaise and other constitutional symptoms.

Traquair [3] reported his studies of 160 cases of toxic amblyopia, including retrobulbar neuritis, and in commenting on the visual defect stated that it was always scotoma in the central area of the field. He stated further:

> When the scotoma is not truly pericentral, but occupies a paracentral area overlapping the fixation area towards one side, it is most commonly the centrocaecal area which is affected. It is seldom that a paracentral scotoma in retrobulbar neuritis occurs on the nasal side of the fixation point in the visual field. There is apparently a tendency to predominant involvement of the crossed as compared with the uncrossed fibers.

The characteristic field defect is a round, central scotoma, from 5 to 15 degrees in size. Peripheral scotomas scattered about the field are not so commonly found, but are as characteristic. When combined with a central scotoma, the peripheral scotoma may be an extension of a centrocecal defect to the periphery, and may vary in size from a small band of dim vision to include any extent of the visual field. The scotomatous defects in some cases are bizarre and changeable during the early part of the disease, particularly in mild forms. Peripheral scotomas appear and disappear, and anopsia changes in amount, although rarely in position. The most constant feature is the central scotoma. Diminished visual acuity and alterations in the visual field may be the only findings on the basis of which a diagnosis of retrobulbar neuritis can be made, and in the absence of discernible changes in the nerve head constitute sufficient grounds for the diagnosis.

The effects of growth of tumors or cysts along the pathways of the nerves are well known. Pressure on the optic chiasm or the optic nerves by expanding tumors or cysts produces characteristic changes in the visual fields. Scotomas, followed by blindness and by atrophy of the nerve, brought about through pressure from the carotid artery or arteriovenous aneurysm adjacent to the cavernous sinus, have been demonstrated.

Much has been written on the anatomic position of the fibers of the optic nerve and their relation to the nasal accessory sinuses, from which one gains the impression that disease of the sinuses or changes in their structural development have a direct bearing on the function of

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2. Friedenwald, J. S.: The Pathology of the Eye, New York, The Macmillan Company, 1929, pp. 24 and 65.

3. Traquair, H. M.: Toxic Amblyopia, Including Retrobulbar Neuritis, Tr. Ophth. Soc. U. Kingdom **50**:351, 1930.

the optic nerves through contiguity. The anatomic variations in the sinuses permit a variety of relationships between the sphenoid and ethmoid cells and the nerve, in its passage through the optic foramen and on to the chiasm. It has been intimated that disease of the mucosa of the accessory sinuses may be transmitted to the optic nerve through direct extension or by diffusion of toxic material along the blood vessels that traverse the region.

The effects on the optic nerve of disease of the nasal accessory sinuses, however, are not established. Many authors take a more or less indecisive position in regard to this relationship. Friedenwald stated that whether there is a causal relationship between these two factors has not been proved. Traquair stated: "In Edinburgh we have come to the conclusion that rhinogenic retrobulbar neuritis is a very rare condition."

De Grósz [4] observed approximately 100 cases of retrobulbar neuritis every year, in 15 per cent of which the cause was nasal. In his opinion:

> The sinus disease connection is obvious—pain, tenderness on pressure, sight reduced, central scotoma—all these symptoms speak for the fact that the disturbance of the accessory sinuses affected the optic nerve. The course of the disease reaffirms our diagnosis, as the application of camomile compresses, the absorption of camomile steam, or salicylates given internally are followed by an improvement which may result in a complete recovery within from two to three weeks. Cure is obtained in these cases (pus) either by plugging the nose for from two to three hours with adrenalin-cocaine mixture, or by clearing away the pathological secretions of the affected mucous membranes.

He again stated:

> Considering that sinusites are frequent, those associated with retrobulbar neuritis are very rare, and it is hard to form a clear comprehension of their relation.
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> At the German Ophthalmological Society, in 1927, Hajek was against operative treatment if rhinologic findings proved to be negative, while Meller stood for it on the basis of mere ophthalmologic examination. Hajek obtained improved vision by means of opening the seemingly normal ethmoid cells of one side, but he observed the same improvement without any surgical intervention when the affection of the other eye set in. Many operated were later found to be due to multiple sclerosis.

Wilmer,[5] in 1930, reported 8 cases of chronic retrobulbar neuritis, 1 of which was attributed to sinus infection. The patients improved after operations on the ethmoid and sphenoid sinuses. Gifford,[5a] in 1931,

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4. de Grósz, Emile: Symposium on Diseases of the Paranasal Sinuses and Relation to Disorders of the Eye: The Etiology of Retrobulbar Optic Neuritis, Tr. Am. Acad. Ophth. and Oto-Laryngol., 1930, p. 21.

5. Wilmer, W. H.: Chronic Retrobulbar Neuritis, Arch. Ophth. **4**:817 (Dec.) 1930.

5a. Gifford, S. R.: Relation of Paranasal Sinuses to Ocular Disorders; Especially to Retrobulbar Neuritis, Arch. Ophth. **5**:276 (Feb.) 1931.

in a paper on the relation of paranasal sinuses to retrobulbar neuritis, referred to many publications in which the percentage of sinus disease was given as a cause of retrobulbar neuritis, and added 13 cases of his own, 4 of which were thought to be due to disease of the nasal sinuses. Nasal origin was considered probable in most of 6 cases of undetermined etiology in his group; the condition of all but 1 patient responded to nasal operations or treatment, with improvement in vision, although in 1 case examination of the nasal sinuses revealed no disease, and in the others no definite sinusitis. He stated: "If optic neuritis were frequently caused by sinusitis, retrobulbar neuritis should develop in a fair portion of cases of sinusitis." He further stated that the condition was caused by purulent sinusitis in about 3.5 per cent of cases. In the 15 or 20 per cent of cases in which no cause can be found, opinions differ as to what percentage is caused by latent or hyperplastic sinusitis. Gifford asserted that there is pathologic evidence that such forms of sinusitis may cause retrobulbar neuritis by direct extension.

Wiener,[6] in 1931, stated:

> Sinus disease is one of the most frequent offenders and may produce either intraocular or retrobulbar neuritis. In the opinion of Sluder, it is not necessary to have pus, but the nerve may be affected in hyperplastic neuritis. I can vouch personally for the truth of this statement.

He stated further:

> I could report case after case where no suppuration was found in the nose, in which optic neuritis or retrobulbar neuritis subsided with shrinking of the mucous membrane of the nose. The visual acuity improves, the scotoma gets smaller or disappears, and the symptoms of the blur are dissipated.

Thomson[7] reviewed recent literature on the subject of conditions of the optic nerve caused by disease of the sinuses, and stated that it is evident that the latent form of sinusitis causing optic neuritis is being rather widely recognized, and operation on the sinuses is being advocated. It is mentioned that the operation is not dangerous when skilfully performed. Thomson stated his belief that disease of the optic nerve is a rare complication among the total number of cases of sinus trouble, but he is also convinced that it is a most serious condition, and one which requires prompt and energetic treatment.

Traquair stated that he had never observed a case of retrobulbar neuritis which he could attribute to a tonsillar infection or a dental abscess, although he had seen cases in which the patients recovered after treatment of these conditions. He said that it was also doubtful

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6. Wiener, Meyer: Optic Neuritis: Etiology and Treatment, Illinois M. J. **59**:219 (March) 1931.

7. Thomson, E. S.: Conditions of the Optic Nerve Caused by Disease of the Sinuses, Arch. Otolaryng. **10**:248 (Sept.) 1929.

whether toxins from these sources have the necessary neurotropic quality to produce retrobulbar neuritis. Letchworth [8] wrote that he had never seen a case in which the origin had been disease of the prostate gland, which, he said, was the most prolific cause of iritis. He expressed the belief that the disease may result from infection about the teeth or tonsils in the alimentary tract, or that pyelitis or infected sinuses, just like any other focus, may account for it, but the sphenoid sinus and possibly the posterior ethmoid sinus may account for it in a different way, owing to their proximity to the nerve. Should a sphenoid sinus be full of pus, the surrounding structures will become edematous, and the nerve in the foramen, where there can be no room for expansion, will be pressed on. He referred to numerous reports of cases of retrobulbar neuritis in many of which roentgenograms revealed unusually small optic foramina. He reported further a case that came under his own observation, that of a boy, aged 16 years, who complained of headaches and complete blindness of the right eye, the sight and fields of the other eye being unaffected. At necropsy, the space surrounding the infundibulum in the interspace between the crura cerebri and the temporosphenoid lobes, and surrounding the chiasma and both optic nerves and tracts, was a mass of infiltration which had extended around the right nerve into the orbit. It was obvious that it was in the optic foramen that the nerve had suffered from pressure, whereas the nerve and tract fibers to the other eye had escaped, although they were surrounded by infiltration.

Of the two reasons that have been advanced for involvement of the optic nerve by infection of the sinuses contiguous to its course, more stress has been laid on the presence of infection in the sinuses or hyperplasia of the sinus mucosa than on pressure on the optic nerve by the walls of the sinus or through constriction of the optic foramen. In spite of the fact that changes in the visual field are not often found in rather extensive diseases of the sinuses, nevertheless in cases of retrobulbar neuritis, rhinologists often are urged to operate on ethmoid and sphenoid sinuses in which they can find no disease.

The manner in which toxic material is absorbed from the sinuses was studied experimentally by Mullin and Ryder.[9] They investigated the lymphatic drainage of the nasal accessory sinuses, and concluded that lymphatic absorption from the antrum, either of bacteria or of inert substances, is by way of the submaxillary and internal jugular nodes. They further concluded that absorption from frontal sinuses seemed to follow the same course as absorption from the antrum. The rate of absorption of toxic materials from the mucosa of the frontal sinuses

8. Letchworth, T. W.: Toxic Amblyopia and Retrobulbar Optic Neuritis, Clin. J. **61**:137 (March 23) 1932.

9. Mullin, W. V., and Ryder, C. L.: Studies on the Lymph Drainage of the Accessory Nasal Sinuses, Laryngoscope **31**:158 (March) 1921.

was studied through experimentation on dogs and cats by Childrey and Essex.[9a] They used histamine, nicotine, ephedrine, epinephrine, rattlesnake venom (crotalin) and phenolsulphonphthalein. Although absorption of certain of these substances occurs from the sinuses, it is invariably much slower than that from subcutaneous tissue. This applies to both normal and infected mucosa. Many times a subcutaneous lethal dose of crotalin was injected into the frontal sinus without producing death of the animal. Furthermore, their experiments indicated that absorption was more rapid through mucous membranes of sinuses that were severely infected and producing pus than through normal mucous membranes.

When one considers the vast number of cases of severe suppurative sinus disease without visual symptoms, the connection between sinus disease and retrobulbar neuritis becomes much less credible. Even in the presence of dehiscences in the bony walls in either acute or chronic disease of the ethmoid and sphenoid cells lying near the optic nerve, the incidence of visual disturbances in patients seen in the Mayo Clinic is negligible. The transmission of inflammation from the sinuses to the optic nerve, either by direct extension, through the blood or lymph stream, or by toxins emanating from slightly thickened mucous membranes and diffused as noxious vapors, has received no convincing experimental corroboration.

The number of microscopic examinations of the optic nerve, made in cases of primary retrobulbar neuritis that have come to necropsy is comparatively small, but degeneration of the nerve fibers from injury, from pressure and from toxic inflammation, gives a very clear picture of the processes that block the function of the nerve in part or in toto.

Degeneration of the axis-cylinders is preceded by changes in the myelin sheath and swelling of the supporting framework of the trunk, compressing the nerve bundles. If the region affected is near the optic disk, the changes can be noted with the ophthalmoscope as swelling and congestion in the nerve and in the nerve fiber layer of the retina. If, however, the situation of the lesion is farther back along the course of the nerve, changes in the nerve head may not be noted with the ophthalmoscope until atrophy has ensued. One characteristic picture of functional blocking of the optic nerve, which I pointed out years ago, is the waxy pallor of the nerve heads, without loss of substance, characteristic of pressure about the chiasm.[10] The nerve heads are pale, but nerve substance can be seen as a translucent material within the scleral ring, obscuring the view of the lamina cribrosa. Restoration of, or improvement in, vision can be prognosticated if the pressure

9a. Childrey, J. H., and Essex, H. E.: Absorption from the Mucosa of the Frontal Sinus, Arch. Otolaryng. **14**:564 (Nov.) 1931.

10. Benedict, W. L.: Early Diagnosis of Pituitary Tumor with Ocular Phenomena, Am. J. Ophth. **3**:571 (Aug.) 1920.

causing the visual disturbance and pallor is removed. The only means that we have of determining whether removal of a tumor of the pituitary gland will improve the vision of a sightless eye is by the ophthalmoscopic appearance of the nerve head. Pressure on the nerve at the chiasm, at the optic foramen or within the orbit will cause blocking of function, and, if it is continued sufficiently long, atrophy of the nerve fibers will result.

Friedenwald stated that atrophy of the optic nerve proceeds in both directions from the injury, although its course is more rapid in the direction away from the retina than toward the retina. He did not state whether the atrophy proceeds more rapidly in the papillomacular bundle of fibers.

Most writers are agreed that multiple sclerosis accounts for the greatest number of cases of retrobulbar neuritis. The pathologic changes in the optic nerve in multiple sclerosis have been described by Dawson.[11] He stated:

> The change in the myelin sheath must be looked upon as the most constant, the most uniform, and in many cases the primary one. In the actual sclerotic areas the unmedullated sheath has entirely perished, but in recent areas and in the advancing peripheral zone of older areas, the changes present themselves in various forms, according to the stages of the degenerative process and according to its greater or lesser intensity.

He distinguished three different stages of pathologic change in multiple sclerosis which agreed with the clinical findings of acute, subacute and chronic stages, and at necropsy he found patches at different stages in their evolution which accorded with the clinical history of the condition, which had usually progressed with remissions and relapses. He could not subscribe to the opinion that multiple sclerosis in itself was due to chronic or acute infection about the sinuses or in other parts of the body where it could be brought to the optic nerves by the blood stream. He found much to favor the view that true disseminated sclerosis is due to a specific, morbid agent which calls forth a clearly defined clinical and anatomic picture, and further, that acute infective diseases, trauma, chill, shock and all known exogenous factors may be exciting factors in lowering the resistance of the organism and then allowing the final determinating factor to operate.

In the foreign literature it appears that multiple sclerosis, as compared to other causes of retrobulbar neuritis, is more prevalent in England and Scotland. For instance, Traquair expressed himself as inclined to believe that about 80 per cent of the cases of retrobulbar neuritis is due to multiple sclerosis, but at the same time he came out in favor of the view that many unexplained cases may be examples of

11. Dawson, J. W.: The Histology of Disseminated Sclerosis, Edinburgh M. J. **17**:229 (Oct.); 311 (Nov.); 377 (Dec.) 1916.

a disease similar to multiple sclerosis but characterized by one lesion only, without the subsequent development of disseminated foci, that is to say, monosymptomatic multiple sclerosis.

Degeneration of the nerve fibers in focal areas is characteristic of multiple sclerosis and of injuries due to pressure. Toxic degeneration, however, more often gives the picture of cellular infiltration with degeneration of the axis-cylinders and atrophy, without the sclerotic thickening found in multiple sclerosis. In other respects the pathologic picture of late inflammation or atrophy of the optic nerve is not characteristic of any etiologic factor.

The etiology of 225 cases of retrobulbar neuritis at the Mayo Clinic was determined as follows: multiple sclerosis in 155 cases, pernicious anemia and nicotine in 14, diabetes in 14, alcohol and tobacco in 28, syphilis in 2, congenital amblyopia in 4, familial causes in 1, sinus disease in 1, postpartum hemorrhage in 1, plumbism in 2 and indeterminate causes in 3.

Of 500 definitely proved cases of multiple sclerosis observed at the Mayo Clinic, disturbance of vision was given as the first symptom in 15 per cent. In an additional 40 per cent, disturbance of vision was a second or third episode. In more than 60 per cent of these cases, intranasal sinus operations had been performed elsewhere because of visual disturbance. In all the cases the syndrome of retrobulbar neuritis was the symptom most commonly observed.

The treatment of retrobulbar neuritis, when the etiology definitely is known, consists in removal of the cause and the employment of such measures as may be expected to restore function to the nerves as quickly as possible. If cases are omitted from consideration that are due to tumors, syphilis, tuberculosis and extraneous toxins, such as alcohol, nicotine and lead, there is a group for which no specific remedy can be employed and in which the causes of the disease cannot quickly be removed. For multiple sclerosis, which accounts for the greater number, there is no recognized adequate treatment. In advanced cases only temporary improvement is noted following treatment, but in early cases, particularly in those in which the visual symptoms are dominant, the response is quick to foreign protein therapy. Treatment should be begun as soon as a diagnosis can be established.

The treatment that I have used with the best success is intravenous injection of triple typhoid vaccine in increasing doses of from 25,000,000 to 450,000,000 bacteria, the injections being given three times weekly, if necessary for from four to six weeks or until it becomes evident that the treatment is of no avail. The therapeutic effect of the vaccine is not directly proportional to the height of the thermal reaction. I have observed, however, that if the reaction to the injection occurs within the first six or eight hours, the therapeutic result is more satisfactory than when the reaction is delayed from twelve to twenty-four

hours. Improvement usually begins with the second injection, and progresses rapidly through the first week or ten days of treatment. A relative central scotoma may remain for another two or three weeks, gradually getting smaller, although often complete recovery of vision is obtained during the first week of treatment. I have not found it necessary to administer additional internal treatment, although at the clinic iodides have frequently been prescribed to be taken for thirty days after vaccines have been discontinued.

In comparing the treatment by means of foreign protein with the effect of operation on the sinuses, it is evident that the improvement obtained is due to the same factor. It has been shown that injection of typhoid vaccine materially increases peripheral circulation. The resultant improvement in circulation of the nerve restores its function. The same effect can be produced by other means, such as the application of a 2 per cent iodine solution to the nasal mucosa, the administration of nitrites or pilocarpine, sweats and other vasodilating agents.

Operation on the nasal sinuses has two effects which have not been fully taken into account by those who advocate them for retrobulbar neuritis. Packing of the nose with cocaine and epinephrine for anesthesia produces, first, ischemia and then congestion of the membranes. Following the operation there is continued congestion of the mucosa of the sinuses and the adjacent tissues until healing is complete. If the operation has been sufficiently extensive, there is commonly a rise of temperature of 1 or 2 degrees F. from the absorption of blood, which, in effect, is autovaccination. These two factors are similar to the effects produced by injections of foreign proteins, and the results are parallel. It is my conviction that drainage of secretion from the paranasal sinuses is not the prime factor in the relief obtained by the operation, but that the hyperemia due to the packing and reaction of the operation and the inoculation by absorption of blood are the factors which are responsible for any improvement noted. This conviction is further supported by the course of many patients following operation. Operations on the sinuses are followed by quick improvement, but too often relapses occur shortly after operation because hyperemia has not continued long enough. By applying a 2 per cent iodine solution to the nasal mucosa or by packing the nose two or three times daily with mild silver protein and allowing the packs to remain in place for three hours, hyperemia can be induced for a longer time, and is reported to be as effective as operations on the sinuses.

Except when it is possible definitely to establish a diagnosis of suppurative disease of the sinuses, I think that advising an operation on the sinuses is unwarranted in any case of retrobulbar neuritis. If a suppurative disease of the sinuses is obviously present, an operation should be performed for relief of the local condition, and additional measures employed to treat the retrobulbar neuritis, for even in the

presence of infection of the sinuses, one cannot be sure that some other factor is not present. Operations on the sinuses, in most instances, probably do little harm. In many cases they do some good. The chief objection lies in the employment of an inadequate and unwarranted procedure, when better methods of treatment are available.

ABSTRACT OF DISCUSSION

DR. STUART L. CRAIG: I think that it would be well to emphasize as a rhinologist the importance of the types which Dr. Benedict has brought out, and the tremendous importance of making, not a superficial, but a complete study of each case. Twenty years ago Dr. Coakley taught rhinologists to realize that it was not always necessary to have pus pouring forth from every cell of the nose in order to have disease of the accessory sinuses. We have undoubtedly operated on patients who did not need it. On the other hand, we have had cases of retrobulbar neuritis which gave such spectacular results that I do not believe one could escape the conclusion that the operation influenced the progress of the disease.

In regard to the pathologic changes which are found in these cases, in my experience it is not the case in which there are free pus and obvious infection which gives the best results. It is the one in which the pathologic changes consist of a roughening of the sphenoid and a thickening of the mucous membrane in the ethmoid or sphenoid cells. I agree with Dr. Benedict that one should not rush into operation without careful deliberation. In the absence of evidence of multiple sclerosis or of tumor I am firmly of the opinion that operation is definitely indicated as a last resort.

DR. FREDERICK M. LAW: In the cases which showed a benefit after an operative procedure, the changes were found in the bony walls, in the bone itself. In a series of cases several years ago, I went into that thoroughly, removed a portion of the bony wall of the sphenoids and ethmoids and sent the specimens to the Pathologic Department. Two different types of disease were reported: (1) an edematous condition following phlebitis, and (2) an arteritis and condensing osteitis. In going back over the roentgenograms of those patients, I found that the condition could be seen and visualized in a stereoscopic film made with attention to the finest details and thoroughly studied. These films particularly must be thoroughly studied with the stereoscope.

All the patients whose condition improved invariably had large sphenoids and large posterior ethmoids. Patients showing small sphenoids and small ethmoids invariably failed to exhibit improvement after operation. The change is apparently circulatory, and if optic neuritis is circulatory in origin there must be some connection between the two conditions. After everything else has failed, and if these particular roentgen characteristics are present, the patient is entitled to an operation on the ethmoids.

DR. ARNOLD KNAPP: I am delighed that Dr. Benedict has come out so flatly and has stated so definitely his position, with which I am absolutely in accord. In searching for the cause of retrobulbar neuritis, I think that we ophthalmologists ought to enlarge our diagnostic horizon beyond the nasal accessory sinuses. To indicate that acute retrobulbar neuritis is not rhinogenic, the following observation has always struck me as pertinent: Why do patients with acute retrobulbar neuritis always go to the ophthalmologist first, and why do we not have these cases referred to us by the rhinologist, who sees so many cases of disease of the ethmoids every day?

DR. MARTIN COHEN: Five years ago Prof. George Weill of Strasbourg read a paper before this section on retrobulbar neuritis. At that time I mentioned in the discussion that I had at one time examined about seventy-five or eighty patients with rhinitis, in which I found that the blind spot was practically normal. I recently saw a case which impressed on me the opposite point of view; it showed not so much retrobulbar neuritis as changes in the disk and macula resembling those in malignant hypertension. The case was that of a young man with ethmoiditis, a septic partial thrombus causing a papilledema of 5 diopters, venous engorgement, a star figure in the macula and sudden loss of vision. An ethmoidectomy and a sphenoidectomy on the right side were followed by gradual improvement in vision. Finally, after six months, the vision in that eye was 20/20. The fundus was normal except for one small focus in the perimacular area.

DR. JAMES W. SMITH: I should like to report on a case which clearly illustrates Dr. Benedict's point that disease of the accessory sinuses is too often stressed as the cause of retrobulbar neuritis. The patient's gait, visual loss and bitemporal pallor immediately suggested the diagnosis of multiple sclerosis, which was confirmed by a neurologist. Six months previously this patient had been seen by an ophthalmologist who made a diagnosis of retrobulbar neuritis. The patient was then examined by a rhinologist who reported sphenoidal and ethmoidal disease. His findings were corroborated by roentgenography of the sinuses. An operation was performed on the sinuses, and was followed by such striking visual recovery that the case was presented before one of the local medical societies as one of retrobulbar neuritis cured by nasal operation. Shortly thereafter the vision failed permanently, and the true diagnosis of multiple sclerosis was clearly established.

DR. SIGMUND AGATSTON: The various types of retrobulbar neuritis may be divided into several groups. There is the very acute type which is definitely associated with an acute sinusitis. These patients get a severe cold with sudden loss of vision, and there the connection is very distinct. Ordinarily shrinkage of the mucous membrane by means of cocaine or mild silver protein will improve the condition rapidly. There should be no discussion about a case of that kind. There are the types caused by multiple sclerosis, the origin of which we do not know. Then there are the toxic amblyopias and another type which is frequently overlooked, one which is caused by circulatory disturbance. The patient will give a history of rapid loss of vision, and apparently there is no change in the fundus. Later vascular changes are seen. In other words, it is possible to have a spasm or a partial occlusion of a branch of the retinal artery going to the macula, which will simulate the symptoms of a regular retrobulbar neuritis.

MAJOR W. F. VON ZELINSKI: It might be of interest to mention that about ten years ago I made a study of about 100 cases of definitely diagnosed sinusitis and a comparative study of 100 other cases in which sinusitis had been excluded. The study consisted of outlining the blind spot to see whether it was enlarged. In the cases not definitely diagnosed as sinusitis, I found a greater percentage of enlarged blind spots than I did in the cases definitely diagnosed as sinusitis.

DR. JOSEPH STANTON HORY: One of the most important ocular findings in multiple sclerosis is the transient scotoma which comes and goes.

DR. WALTER F. DUGGAN: About ten months ago we had a case of unilateral acute retrobulbar neuritis at the Knapp Hospital. The patient had a typical scotoma, and was treated with sweats and injections of a nonspecific milk preparation. After ten days vision was 20/200 in the affected eye and the central scotoma much smaller, but still present. At that time I gave the patient six injections of a compound sodium nitrite solution, and the vision improved to 20/50. Neurologic

examination then gave negative results. Although roentgenograms of the sinuses were negative, a suggestion was made that the sinuses be opened. This was not done, and the patient was discharged. Two weeks later the vision was 20/30 in that eye, and the field had improved. Six and a half months later he came in, and the vision was still normal, the scotoma being smaller. The vision two weeks ago was 20/25. We had at this time a neurologic examination, and the patient showed definite signs of multiple sclerosis. If the sinus had been drained, I am sure that the improvement would have been attributed to the operation. As a matter of fact, the case was one of true multiple sclerosis with an acute unilateral retrobulbar neuritis, which appeared ten months earlier than any other signs of the disease.

DR. W. L. BENEDICT: I wish to make it clear that I have no quarrel with the rhinologists. Really, they are our best friends. They do not operate on healthy sinuses for visual disturbances unless the ophthalmologists tell them to. Recent work on the physiology of the mucous membranes of the sinuses indicates that disease of the sinuses is brought about by disturbances in circulation. From chronic infection or chronic inflammation of the mucous membranes of the sinuses, sclerosis of the tissues and eburnation of the bone result. The vasomotor nerves control the vascularity, and considerable change in the blood supply may be brought about as a result of constitutional disorders. Disease of the sinuses may be brought about by the same condition which causes retrobulbar neuritis. In such a case an operation on the nose could not bring about permanently beneficial effects. Rhinologists do not, as a rule, advise operations on sinuses that have hypertrophic membranes unless such symptoms as headache, lowered visual acuity and vertigo are present. The ophthalmologist schould not be too eager in asking the rhinologist to operate on sinuses unless there is definite local improvement to be expected, but should advise the employment of physiologic restorative methods and medical measures.

INTRACAPSULAR CATARACT EXTRACTIONS BY THE KNAPP METHOD

A REPORT ON ONE HUNDRED CASES

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This report is made with the desire to throw more light on the results of this method of extraction of cataracts. After attempts to perform the intracapsular operation according to the Smith method in the winter of 1928-1929, with rather indifferent results, I was led to undertake the method as advocated by Dr. Arnold Knapp following his report on his third hundred cases before this Section on Ophthalmology in March, 1929.

I decided to keep careful record of all my operations for cataract at Wills Hospital until I could report on one hundred intracapsular operations by his method. This record was completed in February of this year (1932) and embraced operations on one hundred and eighty eyes. The lens was delivered in its capsule in 55.55 per cent of all cases in which operation was attempted between March, 1929, and February, 1932.

PROCEDURE

The pupil was dilated with a drop of atropine solution, which was instilled one hour before operation. Section was usually completed with a small conjunctival flap, followed by iridectomy in the usual manner. If much bleeding occurred, so that the anterior chamber was filled to such an extent as to obscure the view, it was removed by irrigation. The Kalt forceps were then used to grasp the capsule in the lower third, and slow traction was made upward and from side to side until the zonule was broken below and the lower periphery of the lens came into view in the pupillary space and remained there. The Kalt forceps were then released. A curved instrument, such as a Smith hook or a curved spoon, was then grasped in the right hand and pressure made backward at the lower limbus behind the up-tilted lens at the same time that a spoon or spatula in the left hand made pressure against the scleral lip of the incision in a downward direction. In this way the lens was made to tumble, its lower edge being delivered first. It is important that the instrument following the lens upward from below is kept in close contact until the lens is completely delivered, as failure to do this may allow vitreous to come up into the wound. The iris was then replaced and care taken to smooth the conjunctival flap away from the wound edges. Atropine was then instilled, and dressings were applied. This dressing was not disturbed for forty-eight hours.

In the one hundred cases there were eighty mature cortical cataracts, seventeen immature, one nuclear, and two hypermature.

Read before the Section on Ophthalmology, College of Physicians of Philadelphia, Nov. 17, 1932.

COMPLICATIONS

At the time of Operation.—Loss of vitreous occurred in six cases; in one a sudden movement of the eye upward caught the lower flap, and a slight amount of vitreous was lost, but final vision was 6/6 with correction; in another case a small amount of vitreous was lost, which probably would not have affected vision, but on the fourth day the eye was struck, causing intra-ocular hemorrhage, reducing vision to 6/30 with final correction. In four other cases there was a considerable loss of vitreous; one patient finally had 6/9 vision; another had considerable loss of vitreous, but the eye healed promptly, although the patient complained of persistent nausea for about three days. When satisfactory examination could be made, an almost complete detachment of the choroid was seen. This reattached later, and ultimate vision was 6/30 in this eye. A third patient lost a large amount of vitreous by turning the eye upward and catching the flap against the upper lid; final vision in this eye was 6/15 with correction. A fourth patient had a large loss of vitreous from causes not stated in the record, vision of 6/30 with correction being the best obtained. In ninety-four cases there was no loss of vitreous.

After Operation.—Striate keratitis was observed in five cases. This disappeared within a short time, usually before the patient left the hospital.

Iridocyclitis occurred in two cases, from undetermined causes, nullifying the results of operation.

Hyphemia, due to a blow on the eye from four to eight days following operation, occurred in four cases. This cleared without difficulty.

Detachment of the retina was seen in three cases, with consequent loss of vision except for hand movements. I have no adequate explanation for these complications, except for the fact that in one case, at the time of operation, just as the capsule was grasped, the patient made a quick, short excursion of the eye upward, which had the effect of breaking the zonule by a downward movement, which is a movement almost at right angles to that which it is desired to make. In this case, the patient quieted almost immediately, and the completion of the extraction was made by tumbling in the usual manner without further incident. At the first dressing and up to the fifth day the eye looked as though it were healing perfectly, and vision appeared clear to the patient. On the morning of the fifth day, the sight was dimmer, and close examination revealed hemorrhage deep in the vitreous. This eventually cleared, and it was then seen that a retinal detachment had occurred. Vision was lost except for hand movements. I am still undecided as to whether the

movement that has been described caused trauma resulting in detachment, or whether some other factor, such as pressure on the eye during sleep, was responsible.

TIME OF STAY IN THE HOSPITAL FOLLOWING OPERATION

Aside from these complications, recovery was remarkably rapid and free from irritation in many of the cases. In two, the record was not complete. In the remaining ninety-eight cases, of which the date of discharge is known, there were twenty-one patients discharged in seven days, or 21.42 per cent; twenty-four were discharged from the eighth to the tenth day, inclusive, viz., eight on the eighth, seven on the ninth and nine on the tenth day; thirty-eight were discharged from the eleventh to the fifteenth day; the remainder, or fifteen, were discharged at longer intervals.

Stated in another way, of the ninety-eight patients, forty-five, or 45.91 per cent, were discharged within ten days after operation.

Contrasting these figures with those in the cases in which the classic operation was done, it was found that 37.14 per cent of the patients were discharged within ten days following operation. This makes a difference of 8.77 per cent in favor of the group in which intracapsular operation was performed.

VISUAL RESULTS

A study of the visual results in the hundred cases under consideration showed that one patient was a foreigner for whom no interpreter could be obtained through whom he could communicate, and therefore no accurate result could be recorded; that in three cases vision was reduced to hand movements as a result of retinal detachment, and that in two patients no improvement in vision resulted because of iridocyclitis.

In the remaining ninety-four cases:

6/6 vision	was obtained	in 31, or	32.97 per cent
6/9		in 22, or	23.4 per cent
6/12		in 10, or	10.63 per cent
6/15		in 12, or	12.76 per cent
6/21		in 5, or	5.31 per cent
6/30		in 11, or	11.7 per cent
Less than 6/30		in 3, or	3.19 per cent

Expressed in another way, of the ninety-four patients on whom the intracapsular operation was performed, sixty-three, or 67.02 per cent, obtained 6/12 vision or better.

Intracapsular Extractions of Cataract by the Knapp Method

No.	Age	Cataract	Complications	Day of Discharge After Operation	Visual Result
1	53	Mature	Hyphemia, sixth day	11	6/6
2	63	Mature		7	6/6
3	71	Mature		7	6/6
4	66	Mature	Hyphemia, fourth day	14	6/30 on discharge
5	71	Mature		11	6/12
6	55	Immature	Striate keratitis	22	6/30
7	72	Mature	Detached retina	7	Hand movements
8	54	Mature		8	6/6
9	61	Mature	Slight vitreous loss	13	6/6 +9
10	73	Mature	Hyphemia	13	6/15 on discharge
11	70	Mature	Striped keratitis	19	6/15
12	74	Mature	Iridocyclitis, long, low grade	64	Nil
13	76	Mature	Iridocyclitis, low grade	8	Nil
14	65	Mature		7	6/9
15	58	Mature		8	6/6
16	55	Mature	Slight striate keratitis	7	6/15 on discharge
17	60	Mature		7	6/6 —1
18	70	Mature		7	6/9
19	74	Mature		24	6/30
20	57	Hypermature		11	6/6
21	64	Mature		16	6/21 (?)
22	64	Mature		16	6/21 (?)
23	68	Mature		15	6/6
24	68	Mature		9	6/12
25	61	Mature		14	6/6
26	76	Mature		13	6/9
27	52	Mature		8	6/6
28	79	Immature		10	6/6
29	81	Mature		17	6/15 on discharge
30	55	Immature		10	6/15 on discharge
31	71	Mature	Large bead vitreous lost	10	6/9
32	71	Mature		7	6/9
33	72	Hypermature		12	6/6
34	65	Mature	Detached choroid	11	6/30
35	73	Mature		10	6/21
36	65	Mature	Detached retina	12	Hand movements
37	63	Immature		9	6/6
38	65	Mature		7	6/9
39	62	Mature		10	6/6
40	41	Mature		11	6/9
41	41	Mature		8	6/9 +3
42	65	Immature		15	6/9
43	70	Mature		14	6/6
44	70	Mature		14	6/6
45	72	Mature		14	6/30
46	76	Mature		7	6/9 on discharge
47	60	Immature, complicated	Posterior synechia	7	6/60 on discharge
48	67	Mature		8	6/12
49	81	Immature		14	6/12
50	74	Mature		12	6/21 on discharge
51	73	Mature	Striate keratitis	10	6/16 on discharge
52	70	Mature	Moderate loss vitreous	13	6/12 on discharge
53	75	Mature		7	6/15
54	60	Immature		9	6/6
55	66	Mature		9	6/6
56	75	Mature	Large vitreous loss	22	6/15
57	59	Mature		10	6/9
58	67	Mature		17	6/9
59	85	Mature		12	6/40 on discharge
60	75	Mature		21	6/9
61	43	Mature	Hyphemia, fourth day	22	6/9
62	62	Mature		6	6/5
63	70	Mature		14	6/6
64	59	Mature		10	6/9
65	58	Mature		21	6/6
66	45	Immature	Large loss of vitreous	11	6/30
67	68	Immature		7	6/6

Intracapsular Extractions of Cataract by the Knapp Method—Continued

No.	Age	Cataract	Complications	Day of Discharge After Operation	Visual Result
68	68	Mature		8	6/6
69	55	Immature		9	6/6
70	75	Mature		11	6/6
71	70	Mature	Corneal haze; myopic	25	6/15
72	56	Mature		7	6/6
73	72	Mature		12	6/12
74	60	Mature		11	6/30 on discharge
75	70	Mature		12	6/12 on discharge
76	60	Immature		14	6/9
77	73	Mature		12	6/6
78	77	Immature		28	3/60
79	65	Mature		7	6/6
80	62	Immature		7	6/6
81	64	Mature		..	No record; illiterate
82	72	Immature		14	6/21
83	68	Mature		7	6/9
84	71	Mature		14	6/30
85	58	Mature	Striate keratitis	8	6/30
86	58	Mature		11	6/9
87	72	Immature		7	6/6
88	78	Mature		9	6/30
89	72	Mature		11	6/12
90	68	Mature	Small vitreous loss	7	6/12
91	80	Mature		11	6/12
92	79	Mature		18	6/30
93	61	Mature		25	6/9
94	77	Mature		14	6/15
95	74	Mature		12	6/9
96	79	Mature		14	6/15
97	78	Immature		14	6/9
98	80	Nuclear		10	6/9
99	87	Mature		7	6/15
100	68	Mature	Hemorrhage, fifth day; detached retina	9	Hand movements

In sixty-three of the remaining eighty cases of the whole series in which the lens was delivered in the usual manner and of which accurate records are available:

6/6 vision	was obtained in 16,	or 25.39 per cent
6/9	in 15,	or 23.8 per cent
6/12	in 6,	or 9.52 per cent
6/15	in 8,	or 12.69 per cent
6/21	in 5,	or 7.93 per cent
6/30	in 2,	or 3.17 per cent
Less than 6/30	in 11,	or 17.46 per cent

Expressed in another way, of the sixty-three patients in whom the lens was delivered in the usual manner, thirty-seven, or 58.73 per cent, obtained 6/12 vision or better. This also is a difference of 8.29 per cent in favor of the group in which the intracapsular method was used.

The intracapsular operation as developed by Dr. Knapp differs from the classic operation mainly in the use of Kalt forceps in place of the cystotome. It may be attempted in all uncomplicated cases; if unsuccessful, the cystotome or capsule forceps may be resorted to, or, if the capsule tears, the delivery can be accomplished with the same facility as in the classic procedure, with the additional assurance that, as the result of tearing away a large part of the anterior capsule, the resulting secondary operation may be less troublesome.

To me, the recovery in the majority of these cases has been truly remarkable. Many times at the first dressing one could scarcely tell from the reaction that an operation had been performed, and this condition continued to obtain.

From the patient's standpoint, the whole trouble is eliminated at one operation, saving the dread of another operation and the additional expense involved, and at the same time offering one of the best chances of restoration of vision.

My experience thus far has greatly encouraged me to continue using this type of operation, with the hope that with additional experience I may be able to report still better results, as I believe it is the best method of dealing with uncomplicated senile cataract.

308 South Sixteenth Street.

ATROPINE CONTRAINDICATED BOTH BEFORE AND AFTER IRIDENCLEISIS FOR GLAUCOMA

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R. E. Wright,[1] by means of several instillations of atropine after iridencleisis in an eye suffering from simple chronic glaucoma, produced acute congestive glaucoma, which had never before been observed in the affected eye. This observation requires fuller comment than the few lines that I recently published in the ARCHIVES.[2]

Wright [1] wrote:

> The Lagrange method or its modification was adopted in three cases. Stimulated by the excellent late results by Gjessing [3] at Oxford in July last, we once more essayed a trial of iridencleisis (Holth). A number of cases were treated by this method toward the close of the year and a good impression formed of the immediate post-operative results. Both the writer and Dr. K. Koman Nayar, who between them have an operative experience of glaucoma of over 4,000 cases and who have been associated in the treatment of the bulk of these cases, were very favourably impressed, although we did not quite see how the after-treatment, which is an essential part of this procedure, would be applied to the type of patient with which we have to deal. For patients living in the city, readily get-at-able, no doubt it is a simple and apparently harmless method. Unfortunately for our enthusiasm, the first private case in which we employed iridencleisis met with disaster. It was a relatively early case as compared with our usual hospital type, with definite but not extensive field changes, and perfect vision. She never had an acute attack. Iridencleisis was performed in the worse eye; the operation was uneventful—it seemed perfectly good. Atropine was used immediately after the operation and again on the third and the fourth day. On the sixth day, she developed an attack of acute congestive glaucoma. With difficulty—after the most energetic non-operative methods—the tension was reduced to reasonable proportions. The damage was severe, however, and the progress of the eye was continuously difficult. A trephining was eventually performed, but the eye remained a comparative wreck of its former state. No doubt there are objections to trephining, but this experience gave us much food for thought as regards the simpleness and safety of iridencleisis.

Atropine—a valuable prophylactic and remedy against "quiet iritis" after trephining and safely employed until the subconjunctival scleral defect is closed by cicatricial scar tissue—has never been used after iridencleisis for glaucoma in the University Eye Clinic at Oslo or by

1. Wright, R. E.: Report of Government Ophthalmological Hospital, Madras, for the Year 1931, Madras, 1932, p. 10.
2. Holth, S.: Vertical Limbal or Oblique Extralimbal Incision for Iridencleisis in Glaucoma, Arch. Ophth. **8**:489 (Oct.) 1932.
3. Gjessing, Harald G. A.: Holth's Iridencleisis Antiglaucomatosa, Arch. Ophth. **6**:489 (Oct.) 1931.

Dr. Gjessing or myself. Dr. Gjessing [4] said this in plain terms in Helsingfors on June 25, 1931, and in Oxford a fortnight later. He stated that he sometimes "instils 10 per cent cocaine once or twice in order to obtain a suitable mydriasis"; he added "or homatropine, 1 per cent," because he knew that this had sometimes been used—in reality very seldom—both at the University Eye Clinic here and by myself, and never with subsequent acute congestive glaucoma. In reality, Dr. Gjessing instils only cocaine, 10 per cent, which certainly is still safer; I am sorry that this statement (on my advice) was deleted from his manuscript for the ARCHIVES.

Dr. Johan Borthen [5] instils atropine, 1 per cent, ten minutes before his "iridotasis" (a modified iridencleisis) in order to prevent the untouched sphincter muscle from drawing the iris in again. Irritation of the dilatator muscle by atropine, however, can have the same effect, as was frequently demonstrated by the experienced Professor Löwegren [6] in iridial prolapses through traumatic wounds of the corneal periphery. I know of concrete examples of complete retraction of the iris on the same day that the "iridotasis" was done under local anesthesia with cocaine. Borthen, however, never instils atropine after this operation.

It is a misunderstanding to believe that iridencleisis is suitable only for readily followed-up patients in the cities. Neither the University Eye Clinic at Oslo nor I have had an opportunity of seeing patients undergoing iridencleisis as regularly as Dr. Gjessing does, those in Oslo excepted, whom we try to examine every fortnight for weeks or months without the instillation of miotics.

As I myself have done for many years, the University Eye Clinic, since 1930, has prescribed for all country patients the instillation of pilocarpine, 2 per cent, once or twice a day for six months, beginning a fortnight after the iridencleisis; in districts in which pharmacies or dispensing physicians are few and far between, the patients on dismissal from the clinic should obtain one-half ounce (14.2 cc.) bottles of pilocarpine, 2 per cent, which will be sufficient for six months.

Pilocarpine after iridencleisis was not prescribed in the University Eye Clinic between 1920 and 1929. The statistics for these ten years were recently given by Dr. J. C. Holst.[7] The functional results were better after subconjunctival iridencleisis than after anterior sclerectomy.

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4. Gjessing, H. G. A.: Acta ophth. **10**:16, 1932

5. Borthen, Johan: Arch. f. Augenh. **65**:42, 1909; **68**:145, 1911.

6. Löwegren, M. K.: Ögonsjukdomarne, ed. 2, Stockholm, 1900, p. 161.

7. Holst, J. C.: Ergebnisse der Sclerectomia tangentialis (Holth) und Iridencleisis subconjunctivalis (Holth) im Jahrzehnt 1920-1929, Klin. Monatsbl. f. Augenh. **87**:602, 1931.

THE NEW WILLS HOSPITAL

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In celebrating the hundredth anniversary of the founding of the Wills Hospital, it is fitting that the medical administration should report concerning its stewardship and present a chart for the future as it begins the work of the second hundred years.

According to the desire of James Wills, this institution was established and has been operated for the relief of indigent victims of disorders and diseases of the eye. Time does not permit a historic review of the influences that prompted him to found the hospital. By acts of charity during his lifetime and the disposition of his estate, the founder clearly demonstrated his spirit of helpfulness and desire to render practical assistance to the people of Philadelphia and of Pennsylvania who were unable to help themselves. This spirit has prevailed in the minds of those responsible for the execution of the trust.

A survey of the accomplishments of the hospital since it opened its doors for the reception of patients on March 3, 1834, will show the extent to which the surgeons and their assistants have contributed toward translating the purposes of James Wills into actualities.

During those ninety-eight years more than eight hundred thousand patients have been cared for, and in 1931 alone, more than twenty-five thousand were admitted to the clinics and wards of the hospital. In a large percentage of instances these patients, when finally discharged, were either cured of the condition from which they were suffering or greatly improved. In his last testament, James Wills requested that preference be given the citizens of Philadelphia, and although this policy has been carried out implicitly, patients have come to the hospital from nearly every state in the Union. In addition, and as a by-product of great significance, which could hardly have been foreseen by the founder, since 1839 one hundred and fifty-six resident surgeons have received training in the hospital for periods varying from one to one and one-half years. The graduate physicians now living are located in twenty-four states, with two in Canada and one in France, so that the thousands of patients coming under their care have received indirect but nevertheless real benefit from this foundation.

The Wills Hospital was established as an institution limited to the treatment of diseases of the eye, many years before the medical profession recognized that such diseases occupied a special field. It has lived through the period when specialism forced for itself a place in medicine,

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Read at the opening exercises, Nov. 12, 1932.

and it is now living in the era when specialism recognizes that its separate branches cannot function as independent units, but must be closely associated with other divisions in the general field of medicine. Advances in medical science have long since demonstrated the intimate relation between the eye and other organs of the body, and the recognition of this fact carries with it the need for coordination and cooperation with other branches of medicine and with dentistry. To this end, for several years, there has been in operation in this hospital a group of associated clinics headed by men of ability and reputation in their respective fields, to whom are referred patients who require examinations and treatments other than those concerned with the eye itself. In addition, a social service department has been established during the past year. Thus, in a large measure the staff is able to command examinations which formerly had to be referred to the various departments of a general hospital, with the added advantage of coordinated general clinics within our own walls, operated primarily for the benefit of ophthalmic patients. In recent years the facilities of the older established laboratory departments have expanded physically and technically, as far as room and available funds would permit, and, what is more important, there have developed a spirit and enthusiasm among the physicians and assistants in charge of these departments which promise much for the future.

Without going into further details concerning the work that has been done and the aid that has been given during the past century, it may be asserted without reservation that as far as the medical administration of the hospital is concerned the trust has been executed in accordance with the spirit of the founder, and with results which we believe would receive his approval.

When we attempt to visualize the future, our task is made easier by the actual surroundings in which we now find ourselves. This new hospital building should be a symbol of the plane of professional endeavor to which we must attain. The measure of the usefulness of a hospital is not determined only by the amount of actual relief given, but is also concerned with those investigations which contribute to new knowledge regarding the causation and prevention of disease. A community does not derive full benefit from established institutions which fail to encourage research, whether clinical or laboratory, and to supply the facilities and personnel for such work. The service and advantages that our patients are receiving today in respect to more definite knowledge of the causes of many diseases, more prompt and accurate diagnosis and more effective treatment, with a resultant greater conservation of vision, have been made possible through patient, persistent observation and research in the past by many workers in many places. In a large measure, the solution of our present and future problems depends

on a continuance of this painstaking research by the workers of today. Every medical institution, therefore, has an obligation to add progressively to knowledge, as well as to provide for immediate and present-day relief.

The ideal situation in a hospital such as this would be the harmonious cooperation between the clinical staff and a research staff, in which one group would bring the problems of actual disease to the laboratory on the one hand, and the laboratory would contribute new methods and appliances for trial in the clinic and operating room on the other hand. The new Wills Hospital offers a striking opportunity for development in this field. Is it too much to hope that in the near future some friend of ophthalmic science with a spirit of helplessness similar to that which guided James Wills may provide the means by which the hospital will be known for its work in research and preventive medicine, equal to its present reputation for relief and cure?

As we say good-bye to the old and enter this new building with its complete modern equipment, we are impressed with our opportunities and responsibilities. Every means has been supplied for the continuance of the ideas of the founder, and the medical staff is keenly aware of its obligation to carry on the work of the hospital more effectively than before. The Board of Directors of City Trusts and the public may be assured that the devotion and loyalty that have characterized the efforts of those who have given their time and strength and skill during the past hundred years will be continued by the members of the Board of Attending Surgeons and their associates who are now charged with the medical administration of this trust.

WHAT IS WRONG WITH THE APPLICATION OF THE THEORY OF FOCAL INFECTION?

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The letters I have received from some physicians concerning my articles on the subject of focal infection induce me to write on the same question for the third time. The great importance of this subject for the welfare of the population and for the medical profession as well is a good excuse for taking up the valuable time of the reader and drawing his attention to this troublesome question.

WHAT IS WRONG?

A specialist on diseases of the eye, ear, nose and throat of a western city, in a letter to me, said:

> I have for many years been fighting alone with very little success against the terrible epidemic of tonsil snatching by both the general practitioner and the specialist, which I consider the most colossal medical scandal of the century, to say nothing of the sacrifice of millions of teeth.

A physician, a general practitioner, with long experience, who is in charge of a large orphanage in the state of Illinois, thanked me for the satisfaction he had obtained from my paper,[1] and exclaimed: "More power to you!"

An ophthalmologist of Texas wrote:

> When I first glanced at the title[2] of your article, I was inclined not to read it at all, so surfeited have I become with the endless reiteration of focal infections being the cause of this, that or the other thing. . . . It is not an uncommon spectacle to see oculists who on a patient's first visit with some disease of the eye begin immediately making a meticulous search for foci of infection in the nose, throat and mouth before they have made a thorough examination of the eye itself or even made an accurate diagnosis.

As such letters have been written, something must be wrong in this field. What is wrong?

In order to have the moral right to perform operations for elimination of so-called foci of infection, a physician must base this right (1) on scientific experimental proof of the theory of focal infection and (2) on empiric observations in favor of such operations point-

1. Lourie, O. R.: Abuse of the Theory of Focal Infection, Arch. Ophth. **8**:24 (July) 1932.

2. Lourie, O. R.: Focal Infection: An Etiologic Factor in Diseases of the Eye, Arch. Ophth. **5**:893 (June) 1931.

ing to the causal relationship between the foci of infection and the disease.

Most important, of course, is the scientific foundation. This has been laid by Rosenow and, as I tried to show in my last article,[1] does not solve the problem of focal infections. Rosenow's experiments are not convincing, and his conclusions are contradicted by other experimenters.

According to Pette,[3] the teaching of Rosenow that focal infection is the cause of the acute nonsuppurative infections of the central nervous system is in no way supported by the evidence presented by Rosenow himself. This teaching, he contends, is contrary to clinical, anatomic and biologic conceptions based on secure scientific knowledge and should not only be rejected, but combated.

A conscientious physician would not perform operations for elimination of so-called foci of infection merely on the basis of such evidence.

CHANGING VIEWS NECESSITATE CAUTION ON THE PART OF THE PRACTITIONER

The conception of specificity of the causes of infectious diseases has been the basic law of medical science during almost half a century. The fulfilment of Koch's four laws has been considered essential for accepting a micro-organism as the cause of a disease. The newest trend of mind tends to do away with that conception. Rosenow speaks of transmutation, which, in fact, means that the same micro-organism may become the cause of several diseases: A streptococcus of one strain may turn into a streptococcus of another strain; a pneumococcus may turn into a streptococcus. Coburn and Pauli [4] attempt to make Streptococcus haemolyticus responsible for rheumatism, and they base their assertions mainly on serologic grounds. They stated:

> In conclusion, the relationship between the incidence of hemolytic streptococcus and the geographical distribution of rheumatic fever, the relationship between the recrudescence in the rheumatic subject and infection of the throat with hemolytic streptococcus, the development of immune bodies for hemolytic streptococcus at the onset of the rheumatic attack and the apparently specific relationship of antistreptolysin formation to infection with hemolytic streptococcus—together this combined evidence indicates that the infectious agent initiating the rheumatic process is Streptococcus hemolyticus.

Koch, with his long cherished four laws, has become "the forgotten man."

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3. Pette, H.: Die fokale Infektion in ihrer Bedeutung für die Pathogenese organischer Nervenkrankheiten, München. med. Wchnschr. **78**:1981 (Nov. 20) 1931.

4. Coburn, Alvin F., and Pauli, Ruth H.: J. Exper. Med. **56**:676 (Nov. 1) 1932.

On the other hand, Swift,[5] in Cecil's textbook, said:

> Many observers believe the Streptococcus rheumaticus to be the causative agent; more think it is a secondary invader. . . . Our own observations at present indicate that the bacteria occasionally recovered from the blood or nodules are a heterogeneous group of non-hemolytic streptococci, usually called viridans, with no common biochemical or immunological relationship; in several patients we have found two or more distinct strains of viridans and occasionally hemolytic streptococcus in addition. Generally the causative agents of a specific disease fall into one or, at most, a few immunological types. The heterogeneity of the streptococci recovered has forced certain observers to consider rheumatic fever to be a peculiar mode of response on the part of the patient to a number of different types of stimuli, rather than the effect of a uniform stimulus or causative agent. Some even consider it a phenomenon of hypersensitiveness of the type encountered in serum disease. These possibilities are briefly mentioned to emphasize our present uncertainty as to the exact nature of the disease. . . . The immediate causative agent has not been definitely determined; nor has the typical disease been reproduced experimentally by inoculation of laboratory animals.

There seems to be no agreement between Coburn and Pauli on one side and Swift on the other.

The allergic theory tends to show that disease is due to inherited or acquired hypersensitiveness of the body to some agent (bacteria, toxins, proteins, etc.). This conception, fully developed, may lead to the denial of specificity of the cause for disease: Different organisms may react with the same diseases to different causes, or, vice versa, they may react with different diseases to the same causative agent. For instance, eczema can be produced in some people by the application of mercurial preparations; in others, by the application of iodoform. The streptococcus is considered by many to be the cause of scarlet fever, erysipelas and rheumatism (the morphologic and biologic differences of the microorganism may be the result of adaptation). At present it is all in the realm of speculation.

Progress is the antipode of stagnation. The investigations now carried on are fascinating in themselves: They may open new horizons. Physicians are perhaps on the threshold of revaluation of so-called secure scientific truths. If that is so, how is it possible to be sure that the conceptions now arising will not change in the near future? When one reads Zinsser's[6] excellent book "Infection and Resistance," one cannot help knowing how many exceptions to so-called general laws are encountered. These exceptions are so numerous that sometimes it is to be wondered whether the "law itself" is actually a law or an exception. Under such circumstances the practitioner should be cautious in order not to do harm to the patient by the application of treatment based on

5. Swift, Homer F., in Cecil, R. L.: Textbook of Medicine, by American Authors, Philadelphia, W. B. Saunders Company, 1927, pp. 79 and 80.

6. Zinsser, Hans: Infection and Resistance, New York, The Macmillan Company, 1918.

such vague theories, often prematurely proclaimed by their overenthusiastic authors or supporters for infallible truths. Experience teaches that very few of the enthusiastically received methods of treatment survive the severe test of time. Such caution should be exercised all the more since the new theories do not meet with general recognition even among the scientists and experimenters themselves.

CLINICAL OBSERVATIONS ALONE CAN HARDLY PROVE THE CAUSAL RELATIONSHIP BETWEEN FOCAL INFECTION AND OCULAR DISEASE

It is to be regretted that medicine so far is not an exact science. One has to rely on empiric observations for treatment and operations. This is the reason why the advocates of operations for elimination of so-called foci of infection base their claims on observations that show improvement or cure of a disease after such operations.

I have never denied that such improvement takes place in some cases. What I do deny, however, is that the interpretation of these observations is correct in most cases.

"Post hoc, ergo propter hoc" is a wrong way of making correct conclusions. But this mistake is made by many outstanding physicians.

The improvement of an ocular disease after an operation for the removal of so-called foci of infection is usually ascribed as due to the operation without any other proof for the causal relationship between the focus and the ocular disease. In such cases the elimination of all other causes for the ocular disease is insufficient as such a proof because at present it is impossible to eliminate the most important disease—deficient knowledge. One must admit that in many cases the actual cause of the disease remains unknown, however humiliating it may seem to one's intelligence. Many advocates of the theory of focal infection, however, have their minds tuned up in unison with the theory that focal infection is the cause of ocular diseases, and they rush with operations, as though no patient with uveitis or some other ocular disease has ever been improved through local and general treatments. Conservative treatment usually is not given a fair trial.

The following case will illustrate my point of view.

Miss E. Z., aged 23, of New York, a professional dancer, took sick with herpes febrilis corneae of the right eye. She consulted an ophthalmologist in New York, who made a correct diagnosis and advised an immediate tonsillectomy, claiming that the ocular disease was caused by inflamed tonsils. She wired her mother, who happened to live in Boston and to be my patient.

Miss Z. called at my office on Feb. 13, 1932 (the ninth day after the onset). As I do not believe that the exact cause of herpes febrilis is known, I advised to continue local treatment and prescribed acetylsalicylic acid internally. In twelve days no trace of the disease remained, the girl had gained 6 pounds (2.7 Kg.) and she felt perfectly well.

Had the patient undergone a tonsillectomy, I have no doubt that the same results would have been obtained, and an article might have appeared under the title: "Herpes Cornea Febrilis Due to Tonsillitis Cured by Removal of Tonsils."

As I pointed out in one of my previous articles,[2] even the statistics of the advocates of the theory of focal infection show that in the great majority of cases elimination of the foci will be futile. Within the remaining minority, I believe, the largest number belongs in the group of which I have just spoken. All, or almost all, of the numerous patients who would improve under local and general treatment are included in these statistics, because operations are performed promiscuously, those treatments also being used in most cases. How can it be known whether the improvement in such cases is due to local or general treatment, to blood-letting or to elimination of the focus? The answer will be according to the likes or dislikes of the physician. If one excludes the numerous cases in which the patients would improve anyway, there will be little left.

THE THEORY OF FOCAL INFECTION IS WRONG, AT LEAST DOUBTFUL

According to Kaiser,[7] who is rather in favor of the theory of focal infection, in order to save 133 hearts through tonsillectomy (which, in my opinion, is also not proved), one must victimize 19,867 children. The game is not worth the candle.

Gudzent[8a] of Berlin, the author of a book on gout and rheumatism, in an article published in *Therapie der Gegenwart*,[8b] said:

> One must refer more fully to the theory which in the last few years has been advocated in Germany by Pässler and in America by Rosenow, that rheumatism rests on a chronic sepsis provoked by focal lesions (focal infection) in the tonsils, teeth, nose and accessory sinuses and in many other more or less hidden parts of the body, and that a thorough and radical elimination of these lesions leads to a cure of the chronic rheumatism. I do not wish to enter into details regarding my objections to the exaggerated manner in which therapeutic results are ascribed to that theory, but I shall mention that more recent observations could not remove my skepticism. Last year I had the opportunity to study the results of the removal of diseased foci and supposedly diseased foci in twenty carefully selected private patients. In no case have I noticed a lasting result. My dental associate, Levy, could show that in a large series of rheumatic patients only 25 per cent had diseased teeth. On the other hand, my associate, Ruhmann, had at the same time answered the question as to how many patients visiting the dental clinic suffer

7. Kaiser, Albert D.: Incidence of Rheumatism, Chorea and Heart Disease in Tonsillectomized Children: A Control Study, J. A. M. A. **89**:2239 (Dec. 31) 1927.

8. Gudzent, F.: (*a*) Gicht und Rheumatismus, Berlin, Julius Springer, 1928; (*b*) Neuere Ergebnisse der Gicht und Rheumabehandlung, Therap. d. Gegenw. **73**:243 (June) 1932.

from rheumatism. He came to the surprising conclusion that rheumatism occurred only in 12 per cent of the cases.

On the strength of Kaiser's statistics and the just mentioned quotation from Gudzent's article, it is difficult to see how it is possible to make a conclusion that tonsillar or dental lesions are the cause of rheumatism. This confirms my belief that the theory of focal infection is wrong, or at least doubtful, and scientifically is not proved, and that its application, as it is practiced, is harmful to the people.

I have read two aphorisms, one by Claude Bernard and another by J. Proctor Knott, which every physician should bear in mind:

1. When we meet a fact which contradicts a prevailing theory, we must accept the fact and abandon the theory, even when the theory is supported by great names and generally accepted.
2. Take nothing for granted that may seem inconsistent with reason or established facts, simply because someone of acknowledged authority may have said it.

In short, neither general usage nor authoritative names should replace clear reasoning.

As for the miracles that occasionally appear on the pages of medical journals, it seems to me that some physicians use the ways of thinking that are characteristic of the Christian scientist. The latter, however has a distinct advantage: He knows the source of the miracle, while the educated physician with such a weapon as the theory of focal infection cannot account for it. Hysteria and other functional neuroses should not be lost sight of in such "miraculous" cases.

WHOSE IS THE RESPONSIBILITY?

The average physician is too busy to be occupied with the solution of vital problems. He reads the medical journals and takes for granted what the *dii majores* are saying on this or that subject. Physicians with established reputations, leaders in the medical profession, cannot deny some responsibility for the harm brought about by the spread of the theory of focal infection. I happen to know the opinion of some leading oculists that the theory of focal infection has been carried to absurd extremes and in many cases does harm to the people. It is time that the authoritative voices of such leaders should become audible for the common good.

The already mentioned ophthalmologist of Texas wrote:

Where views in the profession such as the ones about focal infection are held with the tenacity of a religious belief and with about as much foundation as certain beliefs, we cannot hope for much change in the near future.

I am more optimistic and think that common sense must prevail before thousands of unnecessary operations will open the eyes of the patients and bring the medical profession into disrepute. As Francis

Bacon has said: "Men mark when they hit and never mark when they miss." For one successful case published, how many unsuccessful or even disastrous cases have never seen the light of publicity? The most precious asset of the physician is the confidence of the patient. One should not play with it.

CONCLUSION

Neither the present state of scientific knowledge nor clinical observations warrant the performance of promiscuous operations for removal of so-called foci of infection in the treatment for ocular or other diseases.

INTERPRETATION OF REFRACTIVE CONDITIONS IN THE PERIPHERAL FIELD OF VISION

A FURTHER STUDY

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AND
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In a former paper[1] an apparatus and method were described for refracting the eye for the peripheral field of vision, and curves were given showing the refractive situation for eighteen cases. It is our purpose in the present paper to make a more comprehensive analysis than was given in that paper of the type of information about the eye that may be obtained by studying the refractive situation from the center to the periphery of the field. Some of this information, so far as we know, can be obtained in no other way of studying the eye that has as yet been described.

The two most important factors in the ametropia for the peripheral field are (1) the effect of the oblique incidence of the rays of light from objects in the peripheral field on the clearness of the image formed, and (2) the effect of size and shape of the eyeball and of anomalies and irregularities in the conformation of the retina on the distance of the percipient elements from the nodal point of the refracting system. With reference to the first of these two factors, it will be remembered that clear images are formed by lenses only when the object to be imaged is located on the principal axis of the lens. When the object is displaced from the principal axis, a distortion of the image occurs which varies in amount with the angle of displacement of the object or with the angle of incidence of the light on the lens. In general, the effect of varying the angle of incidence is that of adding a weak plus sphere and a strong plus cylinder with its axis at right angles to the plane of incidence. The major effect is thus to create a strong astigmatism. A simple formula for this effect in the two meridians may be expressed as follows:[2]

$$F_1 = \frac{F(\mu - 1)}{\mu \cos b - \cos a}$$

$$F_2 = F_1 \cos^2 a$$

From the Research Laboratory of Physiological Optics, Wilmer Ophthalmological Institute, Johns Hopkins Medical School.

1. Ferree, C. E.; Rand, G., and Hardy, C.: Refraction for the Peripheral Field of Vision, Arch. Ophth. **5**:717 (May) 1931.

2. Laurence, L.: General and Practical Optics, London, The Orthos Press, 1908, p. 163; ed. 3, 1920, p. 245.

In these formulas, F represents the focal length of the lens in question; F_1, the focal length in the plane of least refractive power; F_2, the focal length in the plane of greatest refractive power; a, the angle of incidence of the light; b, the angle of refraction at the first surface, and μ, the refractive index of the lens. In both planes, then, the image is brought nearer to the lens in the plane of the oblique incidence and very much nearer in the plane at right angles to it. The refractive effect for the eye would thus be a comparatively small amount of myopia compounded with a strong myopic astigmatism, the amount of both defects varying with the angle of incidence. The effect on vision is modified, however, by the fact that the distance of the retina from the nodal point of the refracting system decreases rapidly as the distance from the fovea is increased. In the greater number of emmetropic eyes the effect of this is to bring the retina between the foci in the two planes or to cause a mixed astigmatism which increases in diopter value as the periphery of the retina is approached.

As a result of the possible combinations of the factors, length of eyeball, shape of eyeball and strength of refracting system, however, many situations may occur. The possible variations in the shape of the eyeball depend on the relation of the length of the polar to the equatorial axis. If these axes are approximately equal in length, the shape of the eyeball is approximately spherical; if the polar axis is longer than the equatorial, the shape tends toward an ellipsoid; and if the equatorial is longer than the polar, the variation in shape is toward an oblate spheroid. If the variation is toward an ellipsoid, a tendency toward hyperopia is exerted in the peripheral field; if toward an oblate spheroid, a tendency toward myopia. The influence of a strong refracting system is toward myopia; also, it tends to increase the difference between the length of focus in the two planes as the periphery is approached, and therefore to increase the astigmatism due to the angle of incidence of the light. The influence of a long eyeball is in general toward myopia and a short eyeball toward hyperopia. Any number of combinations of these factors and of variations in the relative importance of their influence may occur.

The following examples may serve to illustrate some of the possibilities and their effect on the refractive situation in the peripheral field:

1. In an emmetropic eye with a strong refractive system and an eyeball approximately spherical and long enough to render the eye emmetropic for central vision, the focus in one plane would be in front of the retina and in the other behind it, and a strong mixed astigmatism would be found.

2. In an emmetropic eye with a weak refracting system and a correspondingly long eyeball, oval or ellipsoidal, the focus in both planes might fall behind the retina for all or a greater part of the peripheral field and a compound hyperopic astigmatism occur.

3. In an emmetropic eye with a strong refracting system and a proportionately short eyeball of the shape of an oblate spheroid, both foci might fall between the retina and the lens for the more remote parts of the field and a compound myopic astigmatism be found.

4. In hyperopic ametropia with a weak refracting system and an ellipsoidal eyeball, the amount of hyperopia may increase in both planes as the periphery of the field is approached; and in myopic ametropia with a comparatively weak refracting system and an elongated ellipsoidal eyeball, the amount of myopia may decrease in both planes as the periphery of the field is approached. This characteristic influence of the ellipsoidal shape in combination with a weak refracting system toward hyperopia in the peripheral field is doubtless the determinative factor in the eyes classed as type B in our former paper.

5. In hyperopic ametropia with a short eyeball shaped to resemble an oblate spheroid, the defect may change to myopia at some point in the peripheral field. This shape of eyeball rarely if ever occurs in connection with myopia. If it does, the effect is to increase the myopia toward the periphery of the field.

In the foregoing characterizations as to shape, it should be remembered what slight deviations toward an ellipsoid or an oblate spheroid are required to produce several diopters of effect in connection with a refracting system as strong as that which is present in the eye. Such results as we have obtained do not require at all marked variations in the shape of the eyeball for their explanation in terms of the principles which have been noted. From the data we have obtained in a study of the refraction for the peripheral field, certain cases have been selected to illustrate well marked situations that occur. The results for these cases have been plotted in the form of curves. These curves are shown in charts 1 to 4. In the charts, the results for the horizontal plane, the plane of incidence of the light, are plotted as a solid line; for the vertical plane, the plane at right angles to the plane of incidence of the light, as a broken line. Degrees of eccentricity are plotted along the horizontal coordinate and diopters of refractive defect along the vertical coordinate. The condition of no refractive defect is represented by a horizontal line drawn through the center of the chart. Diopters of hyperopia are plotted above this line along the vertical coordinate; diopters of myopia, below it. The diopters of hyperopia and myopia are expressed in terms of corrections needed, plus and

minus, respectively. The value of the interval of Sturm can be read along the vertical coordinate at corresponding points between the solid and the broken lines.

For the interpretation of the curves, the following points may be noted:

1. The relation of both curves to the base line of the chart indicates the type of astigmatism that is present in the peripheral field. If both are above the base line, the astigmatism is compound hyperopic; if both are below, the astigmatism is compound myopic; if one is above and the other is below, a mixed astigmatism is present. The interval between the two curves represents at the various points the diopter value of the astigmatism, the interval of Sturm.

2. The curve drawn as a broken line gives information as to the shape of the eyeball. That is, this curve represents the refraction in the vertical plane at the various points examined, and the refraction in this plane is not strongly affected by the angle at which the examination is made. The distance of the retina at the various points from the nodal system of the refracting system is, therefore, dominantly determinative of the shape of this curve.

3. The breadth of the interval of Sturm, particularly the rate of its increase together with a comparison of the shape of the curves for the horizontal and vertical planes, gives important information as to the strength of the refracting system. This is due to the great effect of the strength of the refracting system on the rate of shortening of the focus for the obliquely incident rays in the horizontal as compared with the vertical plane.

4. A comparison of the breadth of the interval of Sturm in the temporal and nasal halves of the field gives information as to whether there is an asymmetry in the refractive situation for these two halves of the field; and a comparison of the shape of the curves for the horizontal and vertical planes, information as to whether the asymmetry, when present, is caused dominantly by the refracting system, e. g., a tilting of the lens, or by some peculiarity in the conformation of the retina or in the shape of the eyeball.

5. A consideration of the strength of the refracting system in relation to the refractive condition at the center of the field gives information as to the length of the eyeball.

RESULTS

Chart 1 shows approximate emmetropia in four cases—two with mixed astigmatism of different amounts in the peripheral field, one with a compound hyperopic astigmatism and one with a compound myopic astigmatism.

Chart 1 *A* shows a high mixed astigmatism in the peripheral field. An inspection of the curve representing the refraction in the vertical plane indicates an approximately spherical eyeball. The interval of Sturm is broad, but not unusually so, and symmetrical in the two halves of the field; therefore, the refracting system presumably is of medium strength with no asymmetry. Since the eye is emmetropic at the center of the field and the refracting system is of medium strength, the eyeball may be assumed to be of medium length. In this case, therefore, the indications are: a refracting system of medium strength, symmetrically placed with reference to the axis of the eye, an eyeball of

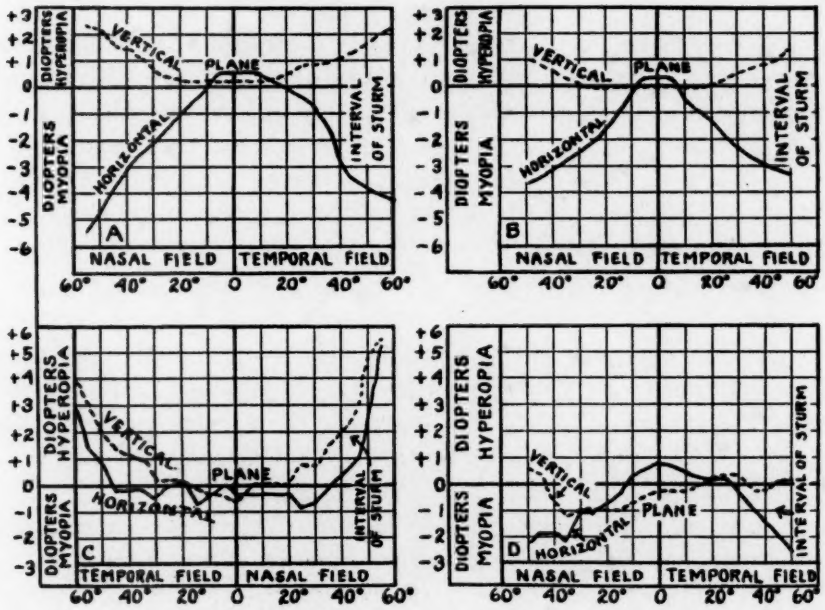

Chart 1.—Four cases of emmetropia, two with mixed astigmatism of different amounts in the peripheral field, one with a compound hyperopic astigmatism and one with a compound myopic astigmatism: *A*, right eye, without cycloplegia, type A; *B*, right eye, with cycloplegia, type A; *C*, left eye, without cycloplegia, type B; *D*, right eye, without cyloplegia, type A.

medium length, approximately spherical in shape, and a symmetrical conformation of the retina.

Chart 1 *B* shows a low mixed astigmatism in the peripheral field. Again, it may be inferred from the curve for the vertical plane that the eyeball is approximately spherical. The interval of Sturm is, however, not so broad as in chart 1 *A*. The indications are, therefore, a weaker refracting system and, since the eye is approximately emmetropic for central vision, a correspondingly longer eyeball. The refraction

seems to be practically symmetrical for both halves of the field; therefore nothing unusual is to be suspected with reference to the placement of the lens and action of the refracting system or in the conformation of the retina and shape of the eyeball.

Chart 1 *C* shows compound hyperopic astigmatism in the peripheral field. The shape of the curve for the vertical plane and the high diopter value of the hyperopia in the far periphery of the field indicate in this case a tendency toward an ellipsoidal eyeball. The interval of Sturm is narrow and decreases in breadth as the periphery of the field is approached. A prominent feature of this case also is the fact that the curve for the horizontal plane is concaved upward in the peripheral field, the pitch of the curve being quite steep in the more remote peripheral portions of the field. This is doubtless a combined effect of weak refracting system and of an ellipsoidal eyeball. A weak refracting system in connection with approximately emmetropic vision at the center of the field indicates an eyeball which is more than usually long. The refractive situation in both halves of the field seems to be practically symmetrical. In terms of the principles stated earlier in the paper, the picture here, then, is that of an emmetropic eye with an elongated, ellipsoidal eyeball, a weak refracting system and practically no refractive asymmetry in the two halves of the field.

Chart 1 *D* represents an eye approximately emmetropic for the center of the field and with a compound myopic astigmatism in the greater part of the peripheral field. The curve for the vertical plane is broad and flat and but little concaved upward. The indication of this is an eyeball flattened at the back, with a shape tending toward that of an oblate spheroid. The curve for the horizontal plane is also comparatively broad and flat and concaved downward, giving in relation to the curve in the vertical plane a representation of compound myopic astigmatism with the rule in the near peripheral field and against the rule in the far peripheral field. The interval of Sturm is rather narrow, indicating a comparatively weak refracting system. It is also fairly symmetrical in the two halves of the field, indicating little if any refractive asymmetry. Since the eye is approximately emmetropic for the center of the field and the refracting system somewhat weak, a rather long eyeball may be inferred. This chart seems to represent an emmetropic eye with a weak refracting system and a correspondingly long eyeball of a shape tending toward that of an oblate spheroid, and with comparatively little refractive asymmetry.

Chart 2 represents two cases of ametropia in which the curves for both the vertical and horizontal planes are concaved upward; that is, the change toward the periphery of the field in both planes is in the

direction of hyperopia. In the first of these, chart 2 *A*, the eye has a low degree of hyperopia in the center of the field which increases quite sharply in both planes toward the periphery, but more sharply in the vertical than in the horizontal plane, thus causing a compound hyperopic astigmatism. The shape of the curve in the vertical plane indicates a somewhat ellipsoidal eyeball. The curve for the horizontal plane follows closely on a broader scale the curve for the vertical plane. The interval of Sturm is narrow, indicating that the refracting system is weak. It is also symmetrical in the two halves of the field. Since the eye is hyperopic at the center of the field and the refracting system is weak, the inference is that the eyeball is short. The important refractive features of this eye, then, may be summarized as follows: simple hyperopia in the central field changing to compound hyperopia toward the periphery of the field; a weak refracting system; a short, ellipsoidal eyeball, and no refractive asymmetry in the two halves of the field.

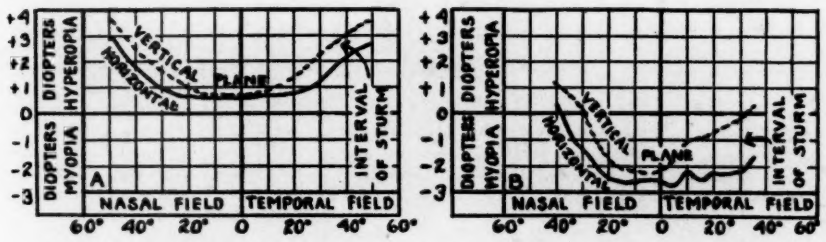

Chart 2.—Two cases of ametropia in which the change in the periphery of the field is in the direction of hyperopia: *A*, right eye, with cycloplegia, type B; *B*, right eye, without cycloplegia, type B.

In the eye represented in chart 2 *B*, a fairly high but unequal degree of myopia is present in both planes at the center of the field. Toward the periphery of the field the myopia decreases sharply in both planes, but the amount of the astigmatism increases. The curve for the vertical plane indicates that the eyeball is quite strongly ellipsoidal. The interval of Sturm is narrow, from which it may be inferred that the refracting system is weak, at least not unduly strong, and that elongation of the eyeball is the determinative factor in the eye's myopic condition. A comparison of the breadth of the interval of Sturm in the two halves of the field shows some refractive asymmetry, and a comparison of the shape of the curves for the horizontal and vertical planes indicates that this asymmetry may be due both to the refracting system and to the conformation of the retina or shape of the eyeball. The refractive features of the eye are, then, compound myopic astigmatism at the center of the field with a change toward a lower value of myopia and

higher value of astigmatism toward the periphery of the field, a weak refracting system, an elongated ellipsoidal eyeball and a comparatively small amount of refractive asymmetry in the two halves of the field.

Chart 3 represents a selection of three cases from a group of cases of ametropia. In *A* is represented an eye which has 2 diopters of myopia at the center of the field and a slight astigmatism. Toward the periphery of the field this changes into a high value of compound myopic astigmatism and finally into a still higher value of mixed astigmatism. The curve for the vertical plane indicates that the eye is strongly ellipsoidal. Toward the far periphery of the field the interval of Sturm is broad, but the shape of the curve for the vertical plane indicates that the shape of the eyeball plays a more important part in this than has been the case in the emmetropic and weakly ametropic eyes which we have thus far been considering. This is, of course, in conformity with the views that are usually held of the more strongly

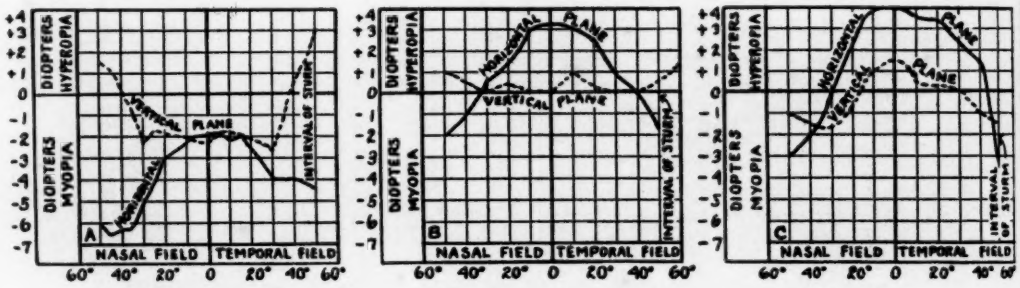

Chart 3.—A miscellaneous selection of three cases of ametropia: *A*, right eye, without cycloplegia, type A; *B*, right eye, with cycloplegia, type A; *C*, right eye, with cycloplegia, type A.

myopic eyes. The refracting system is probably not unduly strong in this case. An elongated eyeball is therefore indicated. Both the breadth of the interval of Sturm and the shape of the curves for the horizontal and vertical planes indicate that some refractive asymmetry is present in the two halves of the field. The chart thus represents an eye which has a simple myopia at the center of the field, a compound myopic astigmatism in the paracentral field and a mixed astigmatism in the far peripheral field; the eyeball is unduly long and ellipsoidal; the refracting system is medium or strong, and there is some refractive asymmetry in the two halves of the field.

Chart 3 *B* represents an eye which is strongly hyperopic in the horizontal plane and emmetropic in the vertical plane at the center of the field, and hyperopic in the vertical plane and myopic in the horizontal plane in the far peripheral field. An analysis and interpretation of the data for such an eye are, of course, difficult to make. That

is, so far as the refractive situation is concerned, an astigmatism represents two types of eye compounded into one. However, the data may contribute something helpful to the better understanding of the eye in question. The shape of the curve for the vertical plane is broad and flat, indicating an eye of the oblate spheroid type. The interval of Sturm is broad, and the curve for the horizontal plane is sharply concaved downward. Since the horizontal is the weaker refracting plane of this eye and there is a strong effect of increase of angle of incidence in this plane, it does not seem probable that the refracting system can be weak. There is little refractive asymmetry in the two halves of the field. The indications are, then, a short eyeball of the oblate spheroid type, a comparatively strong refracting system having less refracting power in the horizontal than in the vertical plane and practically no refractive asymmetry.

Chart 3 *C* represents an eye hyperopic at the center of the field but much more hyperopic in the horizontal than in the vertical plane. Toward the periphery of the field the hyperopia decreases rapidly in both planes but more rapidly in the horizontal than in the vertical plane, changing in turn into a compound myopic astigmatism with the rule and a compound myopic astigmatism against the rule. The curve for the vertical plane is concaved downward instead of upward, as has been the case for all the other eyes which we have as yet had under consideration, and crosses the base or emmetropic line at an eccentricity of about 15 degrees in the nasal field and 30 degrees in the temporal field. That is, from these points on toward the periphery of the field the focus in this plane falls between the retina and the nodal point of the refracting system instead of beyond it. Since the length of the focus is not strongly affected by the angle of incidence in this plane, the change from hyperopia to myopia at these points in the field would seem to indicate for this eye a decided deviation in shape toward an oblate spheroid. The curve for the horizontal plane is sharply concaved downward, and the interval of Sturm is fairly broad. Since the horizontal is the plane of weaker refracting power, this again, as in chart 3 *B*, would seem to indicate that the refracting system cannot be weak. As in chart 3 *B* there is here also little refractive asymmetry in the two halves of the field. The characteristics for this eye seem in general to be very similar to those for the eye represented in chart 3 *B*, namely, a short eyeball, oblate spheroid in type, a fairly strong refracting system and little refractive asymmetry. The deviation in shape toward an oblate spheroid, however, is probably considerably greater than in case of the eye represented in chart 3 *B*.[3]

3. It is interesting to note that 3 *B* and *C* represent the eyes of brothers, 11 and 13 years of age, respectively.

Chart 4 represents cases selected from a group showing a pronounced refractive asymmetry in the two halves of the field. These cases are comparatively infrequent, but they may be found in course of time in any clinic. As was pointed out in the former paper, it is chiefly for the better understanding of, and more intelligent treatment in, these cases that there is a practical need for studying the refractive situation in the peripheral field. In chart 4 *C* the asymmetry may sustain a causal relation to the ocular deviation which was found to be present. In the other two cases the character of the asymmetry is not such as to lead us to suspect any considerable effect on the muscle balance of the two eyes.

Chart 4 *A* represents the superimposition of an asymmetry on an eye which seems otherwise to be of the type shown in charts 1 *A* and *B*.

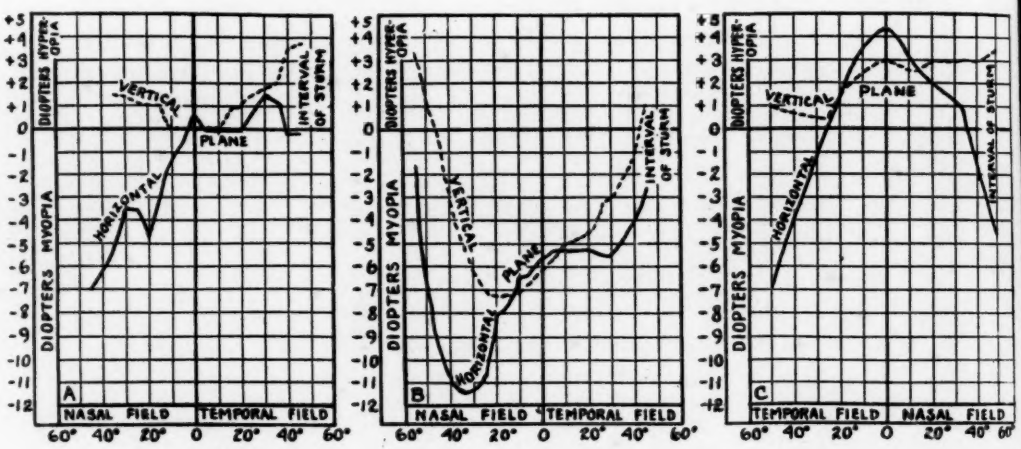

Chart 4.—Three cases having a pronounced refractive asymmetry in the two halves of the peripheral field: *A*, right eye, without cycloplegia, type C; *B*, right eye, without cycloplegia, type C; *C*, left eye, type C, extreme presbyopia, no accommodation.

The curve for the vertical plane is concaved upward and has a breadth and shape that would indicate that the eye is probably of the spherical type. The interval of Sturm is of medium breadth, which would indicate a refracting system of medium strength. Since the eye is emmetropic for the central field, it may be inferred that the eyeball is correspondingly of medium length. A comparison of the breadth of the interval of Sturm for the two halves of the field together with the shape of the curves for the horizontal and vertical planes for these halves of the field indicates that the asymmetry is in all probability due primarily to the refracting system, e. g., to a tilt of the lens toward the temple. This would increase the angle of incidence for the nasal field and decrease it for the temporal field, and cause comparatively

a corresponding amount of shortening of the focus in the nasal field and lengthening of it in the temporal field. Under test, this case showed practically no muscle imbalance. An inspection of the chart shows no reason for suspecting a muscle imbalance, so far as the refractive situation centrally and peripherally is concerned. That is, the eye is nearly emmetropic at the center of the field, and no deviation in either direction could be expected to improve the refractive condition.

In chart 4 *B* is shown a case of high myopia complicated with a pronounced refractive asymmetry in the two halves of the field. The curve for the vertical plane is narrow and strongly concaved upward. Also, the apex of the curve is displaced 20 degrees to the nasal side of the center. The curve would seem to indicate an elongated narrow eyeball, the posterior half of which is strongly ellipsoidal and asymmetrical. The curve for the horizontal plane is also concaved upward with its apex displaced about 35 degrees to the nasal side of the center of the field. At this point the value of the myopia is 11.5 diopters. From here the myopia decreases sharply and becomes 1.5 diopters at 65 degrees. At the center of the field the myopia has a value of 5.5 diopters, and at 45 degrees to the temporal side of the center, 3.5 diopters. The interval of Sturm is comparatively narrow, but broader on the nasal than on the temporal side. The shape of both curves and both the breadth and the shape of the zone representing the interval of Sturm indicate that the shape of the eyeball is the dominant factor in both the myopia and the refractive asymmetry. The test both with the Maddox rod and with prisms showed 10 prism diopters of exophoria. When fixation was taken with the right eye alone, a deviation of 20 degrees toward the temple was shown. Measurements of the cornea showed no asymmetry, and the value of the curvature in both meridians fell within the normal range. Such asymmetry of action as there may have been in the refracting system was probably due, therefore, to the lens, e. g., to a tilt or inclination of the lens toward the temple. The color fields for red and blue, 1 degree stimuli, showed a contraction in the upper half. The field for 1 degree green was very small. The ocular deviation in this case was probably due to the high degree of myopia. Refractive asymmetry apparently had little or nothing to do with it. This may be inferred from the fact that when the myopia at the center of the field was corrected, the deviation practically disappeared and by the fact that a deviation of the eye either temporalward or nasalward could not have been expected to render any effective service in clearing up the defective imagery.

In chart 4 *C* is shown a refractive asymmetry superimposed on an eye of the type represented in chart 3 *B*. At the center of the field there was a high compound hyperopic astigmatism, 3 diopters in the

vertical and 4.5 in the horizontal plane. In the nasal field in the vertical plane there was little change in the hyperopia as far from the center as 50 degrees. In the temporal field it had decreased to 0.5 diopter at 25 degrees and was 1 diopter at 50 degrees. In the nasal field in the horizontal plane it decreased to zero at 35 degrees, and at 50 degrees there was 4.5 diopters of myopia. In the temporal field the hyperopia decreased to zero at 25 degrees, and at 50 degrees there was 7 diopters of myopia. It is significant to note that the nearest approach to emmetropia came at 25 degrees from the center in the nasal field. The defect here was 0.5 diopter of simple hyperopic astigmatism.

The shape of the curve for the vertical plane is broad and flat and tilted downward in the temporal field and slightly upward in the nasal field. It is slightly concaved downward around the center of the field, but not in the more remote periphery. These facts would indicate a shape of eyeball tending toward that of an oblate spheroid, also probably some asymmetry of shape. The curve for the horizontal plane is sharply concaved downward. Also, the interval of Sturm is broad. Since the refracting power in the horizontal plane is 1.5 diopters weaker than in the vertical plane, it can be inferred from the sharpness of the concavity of the horizontal curve downward together with the breadth of the interval of Sturm that the refracting system is comparatively strong; at least it is not weak. Shortness of eyeball is probably, therefore, a strong if not the dominant factor in the hyperopia at the center of the field. The total picture presented by the curves for the horizontal and vertical planes and the zone representing the interval of Sturm indicates that the refractive asymmetry may be due to either shape of eyeball or refracting system or both.

The vision in this eye, the left eye, was 20/200, and could not be improved substantially by correction. The patient stated that prior to fifteen years ago there was, so far as he knew, no vision. An examination for central scotoma gave negative results. That is, while the test object was seen vaguely at the center, it was seen equally clearly there and in the field surrounding the center for 15 to 20 degrees in all directions. A test with the Haitz charts showed that when both eyes were open the vision in this eye was entirely suppressed. When the right eye was closed, however, the figure for the left eye was seen. When both eyes were open there was no observable deviation in either eye; but when the right eye was closed the left immediately turned inward and took a fixation approximately 25 degrees from the median plane. It will be remembered from the chart that the nearest approach to emmetropia in the left eye was at an eccentricity of 25 degrees in the temporal field. When using this eye alone, the patient apparently

turned the eye inward about 25 degrees in order that objects in front should be in the line of least refractive error and the clearest image be formed on the retina.

In the course of the examination of the eye the blind spot was mapped on the tangent screen of the Ferree-Rand perimeter with a 0.5 degree white stimulus on a black background. With the eye turned toward the center of the arc of the perimeter, the fixation object could not be seen with sufficient clearness to give satisfactory control of fixation. In order to secure a correct fixation, the left eye was lined up by means of the telescope inserted in the hollow axle of the perimeter. A large fixation cross (3 cm. in diameter) was moved along the horizontal meridian temporalward to the point at which it could be seen most clearly. The highest visibility was attained at a point approximately 25 degrees from the center of the arc. This, it will be remembered, was the point in the field at which there was the smallest error of refraction, 0.5 diopter simple hyperopic astigmatism. With the fixation object at this point, a fairly satisfactory control of fixation was secured and a map of the blind spot was made.

CONCLUSION

Attention may be called to the following points as incentives for a wider study of the refractive situation in the peripheral field:

1. The possibility of determining both the type and the amount of refractive error at every point in the nasal and temporal halves of the horizontal meridian as far out as 60 degrees by a method that is reasonably feasible, satisfactory and accurate.

2. The important relation which the refractive conditions in the peripheral field sustain to acuity in the peripheral field, to achromatic and chromatic sensitivity in peripheral vision and the limits of the form and color field, and to the anomalies and irregularities in peripheral space perception. In this connection it may be noted that the conditions for the formation of an image are so bad for objects in the peripheral field that one can only marvel that peripheral vision is as good as it is, and that the peripheral portions of the retina should have attained as high a development as they have.

3. The ability to detect and determine the amount of refractive asymmetry in the two halves of the field of vision, and the possibility of determining within rather wide limits in cases of refractive asymmetry whether the defect is in the refracting system and its placement in relation to the axis of vision, or in the conformation of the retina or shape of eyeball, or in both.

4. The possible bearing of asymmetrical refraction in the peripheral field on the explanation of cases in which central vision cannot be substantially improved by correction in eyes which show no central scotoma.

5. The rôle which asymmetrical refraction may play in cases of ocular deviation and the bearing that the demonstration of such a condition may have on treatment.

6. And, finally, the possibility of determining roughly the conformation of the retina and the shape of the posterior half of the eyeball by refracting the eye for the peripheral field; of obtaining information as to the comparative length of eyeball and strength of refracting system in both emmetropic and ametropic eyes, and of estimating with a fair degree of certainty the relative importance of length of eyeball and strength of refracting system as causal factors in the refractive defect in hyperopic and myopic eyes.

EFFECT OF STIMULATION OF POSTERIOR LONGITUDINAL FASCICULUS ON OCULAR MUSCLES

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Although the anatomy of the posterior longitudinal fasciculus has been studied in much detail (see Marburg, Ziehen, Spitzer, Muskens and Grey [1]), knowledge of its function is uncertain on many points. It is known that one-sided injuries of this bundle cause a paralysis of the gaze to this side, which seems to show that each posterior longitudinal fasciculus carries the impulses for conjugate movements of both eyes to its own side (Spitzer [2]). The importance of the posterior longitudinal fasciculus for the conduction of vestibular impulses to the ocular muscles has been doubted by Lorente de Nó,[3] who stated recently that reactions of the ocular muscles supplied by the third and fourth nerves could be produced by stimulation of the labyrinth even if the posterior longitudinal fasciculus was severed on both sides. This result was confirmed by one of us (Spiegel [4]), who furthermore injured the brain stem from the ventral side and showed that extensive bilateral injuries of the formatio reticularis, reaching close to the posterior longitudinal fasciculus, did not prevent the genesis of horizontal as well as of vertical nystagmus produced by labyrinthine stimulation.

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1. Marburg, O.: Entwicklungsgeschichte, makroskopische und mikroskopische Anatomie des Nervus vestibularis, cochlearis und Kleinhirns, in Alexander, G., and Marburg, O.: Handbuch der Neurologie, Berlin, Urban & Schwarzenberg, 1923, vol. 1. Ziehen, T.: Anatomie des Zentralnervensystems, in Bardelebens, K.: Handbuch der Anatomie des Menschen, Jena, Gustav Fischer, 1926, vol. 2, pt. 3. Spitzer, A.: Anatomie und Physiologie der zentralen Bahnen des Vestibularis, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **25**:423, 1924. Muskens, L. J. J.: Konjugierte Deviation von Augen und Kopf bei Hirnstamm-Affektion, Monatschr. f. Psychiat. u. Neurol. **76**:268, 1930. Grey, L. P.: Some Experimental Evidence on the Connections of the Vestibular Mechanism in the Cat, J. Comp. Neurol. **41**:320, 1926.

2. Spitzer, A.: Tumor am Boden der Rautengrube, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **6**:1, 1899.

3. Lorente de Nó, R.: Die Labyrinthreflexe auf die Augenmuskeln, Berlin, Urban & Schwarzenberg, 1928.

4. Spiegel, E. A.: Mechanismus des labyrinthären Nystagmus, Ztschr. f. Hals-, Nasen- u. Ohrenh. **25**:200, 1929.

One has to assume that the posterior longitudinal fasciculus is not the only pathway for the labyrinthine impulses to the nuclei of the ocular muscles, but that a second pathway exists in the formatio reticularis. If this latter system is destroyed, the posterior longitudinal fasciculus alone is still able to conduct vestibular impulses to the ocular muscles. The association of the external rectus muscle of the one side with the internal rectus muscle of the other eye, as observed in conjugate horizontal movements of the eye, is generally explained (see Edinger [5] and Bauer and Leidler [6]) in the following way: The posterior longitudinal fasciculus connects the sixth and third nuclei of the same side, thus innervating the homolateral external rectus muscle, whereas the impulses to the opposite internal rectus reach this muscle by crossing roots of the third nerve. This is in agreement with an experiment of one of us (Dr. Spiegel [4]), who severed the right posterior longitudinal fasciculus in the level of the sixth nucleus and also just behind it, extirpated also the left external ocular muscles, with the exception of the internal rectus, and was then no longer able to produce a reaction of the left eye by rotation of the animal. Recently, however, Klossowsky and Levikowa [7] published reports of experiments in which they severed the posterior longitudinal fasciculus on one side in the cranial part of the pons, extirpated on both sides the external rectus and the external bundle of the musculus retractor bulbi and found that after these operations the nystagmus disappeared in the homolateral eye and was weaker in the contralateral eye. They concluded that the internal rectus is innervated in horizontal nystagmus by fibers from the homolateral third nucleus and explained the aforementioned experiments of Spiegel by the assumption that in this experiment crossing fibers from the nucleus triangularis into the opposite posterior longitudinal fasciculus were injured. If their experiments and theories are correct, the posterior longitudinal fasciculus should carry in the cranial part of the pons ascending fibers to the homolateral internal rectus. A more detailed study of the protocols in this article reveals that in quite a number of experiments the rotation of the cats was unable to produce a nystagmus, either on the side of the severance of the posterior longitudinal fasciculus or on the other eye, which phenomenon is not explained by their theory. Hence, it does not seem possible to draw definite conclusions from these experiments.

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5. Edinger, L.: Vorlesungen über den Bau der nervösen Centralorgane des Menschen und der Tiere für Aerzte und Studirende, ed. 8, Leipzig, F. C. W. Vogel, 1911.

6. Bauer, J., and Leidler, R.: Ueber den Einfluss der Ausschaltung verschiedener Hirnabschnitte auf die vestibulaeren Augenreflexe, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **19**:219, 1912.

7. Klossowsky, B., and Levikowa, A. M.: Der Mechanismus des vestibulären Nystagmus, Arch. f. d. ges. Physiol. **228**:198, 1931.

One sees that the conduction of impulses through the posterior longitudinal fasciculus in the innervation of horizontal movements of the eye is questionable; but the relation of this system to the innervation of vertical movements of the eye is still more hypothetic. One knows that lesions in the neighborhood of the aqueduct or in the cranial part of the pons may produce paralysis of the gaze in the vertical direction (Spiller [8]). On the other hand, Poetzl and Sittig [9] assumed that lesions of the ventrocaudal part of Deiters' nucleus are able to produce a skew deviation. Recently, Marburg [10] observed a combination of paralysis of the gaze to the left side and upward in a case of glioma of the midbrain; he conjectured that the left posterior longitudinal fasciculus carries the impulse for the gaze not only to the left side, but also upward, and that the right posterior longitudinal fasciculus carries the impulses for movements of the eyes to the right and downward; however, further observations seem necessary to substantiate this theory.

OBSERVATIONS

Considering the aforementioned problems, it seemed worth while to study the effect of circumscribed stimulation of the posterior longitudinal fasciculus on the eyes.

The experiments were performed on cats. Under ether narcosis, both carotid arteries were clamped, the skull was opened on both sides, and the forebrain and interbrain were extirpated, with the exception of the most ventral parts, to avoid an injury to the nerves of the ocular muscles. A lesion of the corpora quadrigemina was also carefully avoided. Then the narcosis was discontinued, the tentorium was removed, the underlying dura was opened, and the lobus anterior cerebelli was slowly separated from the colliculus inferior by small pledgets of cotton wool, and elevated from the anterior floor of the fourth ventricle. In further experiments, not only the cranial part of the fossa rhomboidalis but the whole floor of the fourth ventricle was exposed by a total extirpation of the cerebellum after removing the occipital bone with the membrana atlanto-occipitalis and also carefully separating the posterior part of the cerebellum from the fossa rhomboidalis. In this procedure it is important to prevent pulling on the brachia cerebelli to avoid trouble in the function of the respiratory center. The diffuse electrode was fastened on the abdomen; the stimulation was made by Sherrington's single wire electrode. Only quite weak faradic currents were used, just sufficient to produce a slight contraction if applied to an exposed muscle. In the intervals between the stimulations, a pad of cotton wool soaked with warm Ringer's solution was put on the fossa rhomboidalis to protect it from drying and cooling. Before each stimulation, the area

8. Spiller, W.: Paralysis of Upward Associated Movements, Arb. a. d neurol. Inst. a. d. Wien. Univ. **15**:352, 1907; Am. J. M. Sc. **158**:695, 1919.

9. Poetzl, O., and Sittig, O.: Klinische Befunde mit Hertwig-Magendischer Augeneinstellung, Ztschr. f. d. ges. Neurol. u. Psychiat. **95**:701, 1925.

10. Marburg, O.: Probleme der vertikalen Blickbewegung, Ztschr. f. Augenh. **58**:253, 1926.

was wiped dry to avoid spreading of the current. To observe the movements of the eyes, the membrana nictitans was removed, the palpebral fissure was enlarged by an incision on the external canthus, and the eyelids were separated by eye specula.

Muskens,[11] who has made excellent researches on this system, thinks it still unproved that the posterior longitudinal fasciculus is electrically excitable in its course between the sixth nucleus and the posterior commissure. It should, therefore, first be stated that the posterior longitudinal fasciculus is excitable by the faradic current. One must surely avoid mistakes due to an escape of the current to the nuclei or roots of the cranial nerves, to systems of the formatio reticularis or to fibers from the flocculus [12] to the oculomotor nuclei (Klimoff-Wallenberg fibers). One could, therefore, use only the observations in which movements of the eye were elicited by minimal currents and only from points just adjacent to the midline, but not from lateral parts of the fossa rhomboidalis near the brachium conjunctivum (which the Klimoff-Wallenberg fibers join). Furthermore, control experiments were made in which the stimulation of the posterior longitudinal fasciculus was compared with that of the fourth nerve, of the corpora quadrigemina and of the lateral part of the floor of the fourth ventricle (see cats Sc. XI and Sc. XII).

On May 24, 1932, the fourth ventricle of cat Sc. XI was exposed, with the following response to tests:

Cat Sc. XI

	Stimulation	Effect on Left Eye	Effect on Right Eye
11:50	Distance of coils 12 cm., right posterior longitudinal fasciculus	Slightly to right	To right
11:55	Same	Slightly to right	To right
12:00	Same current, right fourth nerve.....	.....	Rotation with upper part of cornea inward

Finally, in some experiments, a transverse incision was made through both posterior longitudinal fasciculi in the cranial part of the fossa rhomboidalis, and the stimulation was repeated on points behind the incision (see cat Sc. IV).

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11. Muskens, L. J.: Konjugierte Deviation von Augen und Kopf bei Hirnstamm-Affektion, Monatschr. f. Psychiat. u. Neurol. **76**:279, 1930.

12. Dusser de Barenne (in Alexander, G., and Marburg, O.: Handbuch der Neurologie, Berlin, Urban & Schwarzenberg, 1923, vol. 1, p. 633) mentioned that he was unable to produce movements of the eyes by stimulation of the cerebellum in the cat. We found that stimulation of the cortex cerebelli and even of the flocculus had no effect on the ocular muscles, although distinct movements of the eyes could be elicited in the same animal and with the same current by the stimulation of the posterior longitudinal fasciculus.

On Feb. 2, 1932, the anterior part of the fossa rhomboidalis of cat Sc. IV was exposed, with the following response to tests:

Cat Sc. IV

	Stimulation	Effect on Left Eye	Effect on Right Eye
12:40	Right posterior longitudinal fasciculus in the cranial part of the pons	Downward	Upward
12:52	Left posterior longitudinal fasciculus.....	.....	To left
12:53	Right posterior longitudinal fasciculus.....	Downward after removal of the electrode	First upward then downward (after removal of the electrode)
1:00	Right posterior longitudinal fasciculus.....	Slightly upward	First upward, then downward after removal of the electrode
1:02	Puncture of the left posterior longitudinal fasciculus	Upward	Upward
	Second puncture of this bundle on left side in the cranial part of the pons	.....	To left
1:05	Right posterior longitudinal fasciculus.....	.....	Upward
	Left posterior longitudinal fasciculus behind the puncture	No sure reaction (slight undulation)	
1:10	Puncture of the right posterior longitudinal fasciculus	Downward	Downward
1:12	Right posterior longitudinal fasciculus behind the puncture	No reaction	
1:15	Right posterior longitudinal fasciculus behind the puncture	.....	Slightly upward
1:16	Transverse section through both posterior longitudinal fasciculi behind the punctures	.....	Upward
1:20 to 1:26	Repeated stimulation behind the transverse section	No eye reactions	
		(Respiration still regular)	

Histologic examination of the brain stem on serial sections showed an injury to the floor of the fourth ventricle in the midline and above the right fasciculus longitudinalis posterior, starting in the level of the sixth nucleus. This lesion increased in the cranial sections; finally both posterior longitudinal fasciculi were destroyed in the level of the nucleus motorius trigemini. Just cranially from this lesion a puncture of the left posterior longitudinal fasciculus was found, which ended ventromedially from this bundle in the midline. In the transition of the pons to the midbrain the left posterior longitudinal fasciculus was punctured a second time; this puncture injured, in more caudal sections, the middle parts, in more cranial sections, the medial parts of this bundle. On the right side, the lesion started in the most cranial parts of the pons outside the posterior longitudinal bundle; in the next sections, at the beginning of the corpus quadrigeminum posterius, it reached the right posterior longitudinal fasciculus, which was destroyed in its external and middle parts; its innermost bundles were intact (fig.).

The effects produced by the unipolar stimulation of the posterior longitudinal fasciculus in the cranial part of the pons were not limited to the ipsilateral eye, but were also noticeable in the opposite eye. One could object that this was due to a spread of the current to the opposite posterior longitudinal fasciculus; therefore, it was studied whether the effect on the opposite eye disappears after severance of the nonstimulated posterior longitudinal fasciculus cephalad to the place of stimulation. It was found that the effect on the eye on the nonstimulated side can still be elicited (see cat Sc. VIII).

On April 16, 1932, decerebration cephalad to the midbrain and extirpation of the whole cerebellum were performed on cat Sc. VIII, with the following response to stimulation:

Cat Sc. VIII

	Stimulation	Effect on Left Eye	Effect on Right Eye
12:33	Left posterior longitudinal fasciculus; distance of coils 12 cm., 45°	Upward	
12:37	Right posterior longitudinal fasciculus.....	To right	First fast undulation; after stimulus stopped: downward
12:39	Right posterior longitudinal fasciculus..... Incision into left posterior longitudinal fasciculus in cranial part of the pons	To right	To right
12:40	Right posterior longitudinal fasciculus behind incision (12 cm. distance of coils)	To right	To right
12:44	Right posterior longitudinal fasciculus behind incision (12 cm. distance of coils, 45°)	To right	To right
12:48	Same	Upward	To right
1:05	Same	.....	To right
1:17	Same	Upward	Upward

Beginning of the corpus quadrigeminum posterius (cat Sc IV). *A* indicates the aqueduct; *R*, raphe; *ll*, left lesion; *rl*, right lesion; *lplf* (*rplf*), left (right) posterior longitudinal fasciculus.

Also, a mechanical stimulation of the posterior longitudinal fasciculus in the cranial part of the pons (by puncture, cat Sc. IV) was able to produce a deviation of the opposite eye toward the side of stimulation.

It seems, therefore, correct to assume that ascending fibers of the posterior longitudinal fasciculus send impulses to the internal rectus muscle of the opposite side. But it should be emphasized that this reaction of the opposite eye is found only if the preparation is in good condition, and that it usually disappears after repeated stimulation.

A simultaneous reaction of the homolateral eye is sometimes missed (cats Sc. III and Sc. IV); in other cases (see the protocol of cat VIII), an abduction of this eye, in other words, a conjugate movement of both eyes toward the stimulation, can be observed, even if one stimulates the posterior longitudinal fasciculus cranial from the sixth nucleus. This effect on the homolateral eye could be due simply to a spread of the current to the sixth nucleus, but this possibility could be excluded as repetition of the stimulation after lesion of the posterior longitudinal fasciculus behind the stimulated zone was no more effective (see cat Sc. XII).

On May 24, 1932, the fourth ventricle of cat Sc. XII was exposed, with the following response to stimulation:

Cat Sc. XII

	Stimulation	Effect on Left Eye	Effect on Right Eye
4:20	Left posterior longitudinal fasciculus in the cranial part of the pons (distance of coils 12 cm., 60°)	Outward	
4:22	Incision between the point of stimulation and the area of sixth nucleus		
	Stimulation repeated cephalad to incision...	No effect	No effect
4:30	Right posterior longitudinal fasciculus.....	.....	Up and down undulation
	Lateral part of the fourth ventricle near brachium conjunctivum	.....	No effect, or only a very slight movement of the eye

Stimulation of descending fibers of the posterior longitudinal fasciculus is quite possible, but another possibility also must be considered. As nerve fibers are able to conduct an impulse in both directions, even in a stimulation of ascending fibers of the posterior longitudinal fasciculus, a part of the impulse could be conducted in a caudal (antidrom) direction to reach finally the sixth nucleus by collaterals of the posterior longitudinal fasciculus.

It is interesting that stimulation of the posterior longitudinal fasciculus did not always produce associated movements of the eyes in the same direction. Besides the rare observation of a skew deviation (cat Sc. IV), another form of dissociated movements of the eye was observed (cat Sc. V).

On March 5, 1932, the fossa rhomboidalis of cat Sc. V was exposed, with the following results of tests:

Cat Sc. V

	Stimulation	Effect on Right Eye	Effect on Left Eye
12:15	Left posterior longitudinal fasciculus (distance of coils 11 cm.)	To left	
12:16	Same	To left	Vertical undulation

Sometimes the reaction on the homolateral eye begins with an upward movement, is followed by an undulation, and finishes by a downward movement when the stimulation is discontinued. How can one explain such cases in which the stimulation of the posterior longitudinal fasciculus elicited a horizontal movement toward the side of stimulation on the opposite eye and a vertical movement on the homolateral eye? It is known through the experiments of Bernheimer [13] on monkeys that in the oculomotor nucleus the center of the internal rectus is near the center of the superior rectus; the experiments of Hensen and Voelckers [14] on dogs lead to a similar conclusion. Hence, one must expect that also in the posterior longitudinal fasciculus the fibers ascending to the nuclear centers of these two muscles are very near each other, and, therefore, can be stimulated at the same time. As the superior rectus receives its impulses from the homolateral nucleus, whereas the internal rectus gets the impulses carried by the posterior longitudinal fasciculus through the crossed roots of the third nerve, the production of vertical movements on the side of the stimulation of the posterior longitudinal fasciculus and of horizontal inward movements on the other side can be explained without much difficulty.

Special care was taken to determine whether relations of the posterior longitudinal fasciculus on a certain side to upward or downward movements could be found. But we were unable to discover here any rule, although upward movements as well as downward movements could be observed after stimulation of the posterior longitudinal fasciculus in the cranial part of the pons. It might be remarked that such vertical movements were particularly observed after repeated stimulation of the posterior longitudinal fasciculus, the fibers carrying impulses for horizontal movements apparently losing their excitability usually earlier than those for vertical movements.

SUMMARY

1. Attempt was made to stimulate the posterior longitudinal fasciculus in the cranial part of the pons by unipolar faradic stimulation. Horizontal as well as vertical movements of the eyes could be observed.

2. These experiments support the assumption that the posterior longitudinal fasciculus carries ascending impulses to the crossed internal rectus muscle.

3. Besides movements of both eyes in the same direction, dissociated movements of the eyes were also observed, particularly horizontal movements of the opposite and vertical movements of the homolateral eye.

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13. Bernheimer, S.: Experimentelle Studien zur Kenntnis der Innervation der äusseren vom Oculomotorius versorgten Muskeln des Auges, Arch. f. Ophth. **44**:481, 1897

14. Hensen and Voelckers: Ueber den Ursprung der Akkomodationsnerven, Arch. f. Ophth. **24**:1, 1878.

RETINITIS JUXTAPAPILLARIS

REPORT OF A CASE

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The case to be reported belongs to a group that was originally described by Jensen [1] in 1908, and diagnosed by him as retinochoroiditis juxtapapillaris. Since then more than thirty cases have been reported in the literature; no doubt many more have been observed. The striking transitional character of the retinal lesion and the light it throws on the etiology of the disease make the case both interesting and instructive.

REPORT OF A CASE

History.—A girl, aged 17, apparently well developed, came to the Herman Knapp Memorial Eye Hospital on Aug. 15, 1932, complaining of blurred vision in the left eye over a period of two weeks. About the last week in July she had suffered from a cold in the head and at the same time had noted a dull ache in the left eye on motion. On the following day the ache disappeared, and the patient noted a constant blur before the left eye. There was no history of any previous ocular disturbance, and the past medical and family histories were unimportant.

Examination.—Vision in the right eye was 20/20; the eye was normal. The field was normal. Vision in the left eye was 20/50. The cornea appeared clear and did not show the presence of corneal deposits. The anterior chamber was of normal depth. The iris was normal. The pupil reacted well. The fundus presented a picture of what appeared to be a choked disk of at least 4 or 6 diopters, and a vitreous filled with fine dustlike opacities. Closer examination showed that the disk was almost completely covered with an exudate, with the exception of a small area on the margin between 1 and 2 o'clock. The exudate was evenly spread over an area of at least 2 disk diameters and was covered with many large hemorrhages. The retinal vessels were markedly engorged and tortuous. The macular region was edematous and presented a fan-shaped figure in the macula lying toward the disk. In the lower nasal quadrant and toward the periphery there were numerous superficial, discrete, yellowish-white areas, the largest being slightly larger than pinhead in size. These areas were not associated with any pigmentary changes and were thought to be atrophic points in the retina probably due to the decrease in the nutrition of the retina.

Fields taken at this time showed a sector defect in the upper temporal quadrant which included the blind spot and extended to the periphery. A little of the adjoining nasal field was involved.

The patient was admitted to the hospital for study. Physical examination gave negative results. Roentgenograms of the teeth showed dental caries, but no

1. Jensen, E.: Retinochoroiditis juxtapapillaris, Arch. f. Ophth. **69**:41, 1908.

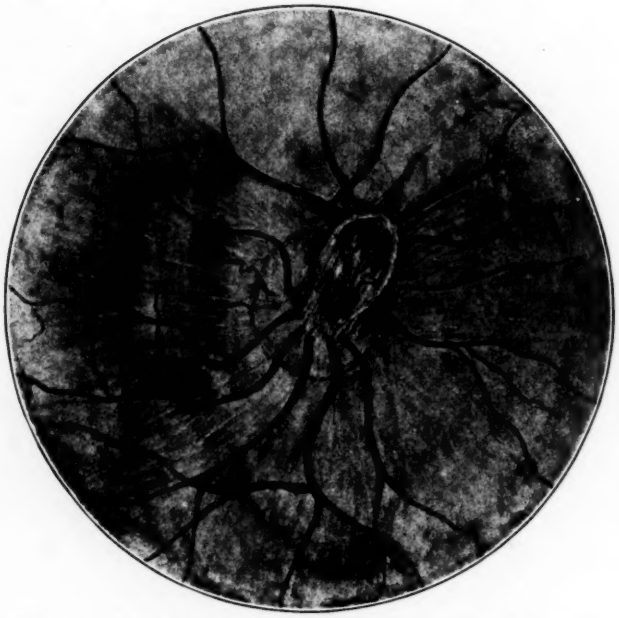

Fig. 1.—Drawing of fundus four months after onset.

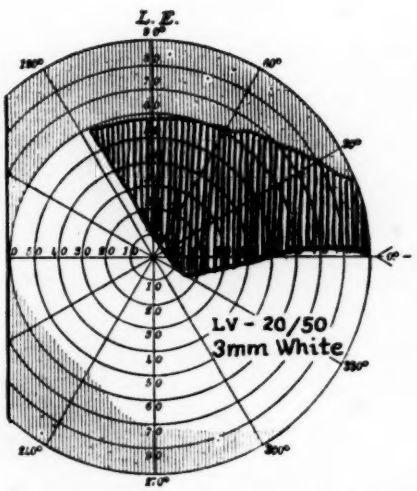

Fig. 2.—Visual field, taken on Aug. 15, 1932.

evidence of an infection. A roentgenogram of the chest showed no signs of any new or old tuberculous process. The Wassermann reaction was negative. Examination of the nose and throat showed the presence of a mild chronic tonsillitis which was not considered to be of any etiologic importance. Roentgenograms of the sinuses showed a cloudy left antrum, but nothing pathologic was found when the antrum was washed out. The Mantoux intradermal test and the subcutaneous test with new tuberculin gave no reaction whatever.

The condition of the fundus improved slowly. Central vision remained practically the same. The tremendous exudate lying on the disk gradually flattened out, and the star-shaped figure in the macula disappeared, but the edema remained. The only improvement noted in the field was that the sector defect was confined to the upper temporal quadrant.

After the edematous opacification of the retina had subsided altogether, a process that took about three months, the details of the fundus could be made out clearly. The surface of the disk appeared slightly congested. On the nasal margin, extending from 5:30 o'clock to about 11 o'clock, there was a crescent-shaped, grayish area that was elevated 1 or 2 diopters. The widest part of this crescent measured one-third disk diameter. The grayish area seemed to lie in the nerve fiber layer of the retina and was not surrounded by any pigmentary changes of the pigment epithelium or any atrophic changes of the choroid. There was an anomalous distribution of blood vessels on the surface of the disk. No physiologic cup could be made out, and in its place were noted many fine capillary hemorrhages. Extending into the vitreous from the center of the disk there was a fine, wavy, veil-like, black substance. The veins passing over the disk were slightly engorged, and the caliber of the arteries seemed to be reduced in size, especially in the lower nasal quadrant. The inferior nasal veins and arteries passed beneath the grayish area described. In the lower nasal quadrant one could still note the discrete yellowish-white areas previously described.

COMMENT

The original description of the disease, given briefly, is as follows: "The appearance of a cotton wool white patch near the papilla surrounded by either atrophic changes in the choroid or a disturbance in the pigment epithelium, and associated with a corresponding sector defect in the visual field that extends to the periphery and includes the blind spot." When the disease comes on acutely, as was noted in the case reported, the entire picture may at first be obscured by what appears to be a severe optic neuritis, but after the edematous opacification of the retina has subsided the true character of the disease can be readily noted. Pallarés [2] described a case of neurofibrillitis tuberculosa retinae which resembled my case a great deal in that the picture of the fundus was about the same in the late stage of the disease. Both cases had the cotton wool white patch near the border of the disk which was not associated with any choroidal or pigmentary changes. In all the other cases of retinochoroiditis juxtapapillaris that have been

2. Pallarés, J.: Neurofibrillitis tuberculosa retinae, Klin. Monatsbl. f. Augenh. **86**:598, 1931.

described there were atrophic changes in the choroid and pigmentary changes in the pigment epithelium. The location of the primary lesion in the case described and in the one recorded by Pallarés must have been in the anterior layers of the retina, and the process did not extend to the choroid. Groes-Petersen [3] and van der Hoeve [4] have cited cases in which the pathologic changes were explained on the basis of a neurofibrillitis retina caused by a cellular infiltration of the anterior layers of the retina. Jensen maintained that the primary lesion is in the choroid and that the neuro-epithelium is involved by direct extension. I think that he is correct in those cases in which atrophic changes in the choroid and a disturbance of the pigment epithelium can be demonstrated. Also it is possible that the primary focus can be in the anterior layers of the retina, and that it can spread to the choroid directly. I have not been able to determine exactly whether it was Groes-Petersen or van der Hoeve who first described the disease as a neurofibrillitis retinae. While it is true that the nerve fiber layer of the retina is affected, the diagnosis of the disease as neurofibrillitis retinae would in my estimation be incorrect, since I have always associated neurofibrils with nerve fiber endings and not with the nerve fiber. The case was examined by Dr. Arnold Knapp, and he suggested the diagnosis of retinitis juxtapapillaris.

The condition usually affects young healthy adults with no evidence of syphilis or any constitutional disease. According to the description of the disease in "Kurzes Handbuch der Ophthalmologie," [5] all of the etiologic factors that are concerned in the production of any acute chorioretinitis are to be considered in the etiology of this disease. Among the possible causes are mentioned syphilis, grip, tuberculosis and even multiple sclerosis.

Abraham [6] has recently published a review of the literature with a description of two cases. The fields that are used to demonstrate the second case are not typical of the disease, and on this account the probability of an exudative retinitis should be given more thought.

According to Dimmer,[7] the pathologic changes in the disease are similar to those in any choroiditis with, of course, the difference in location of the lesion.

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3. Groes-Petersen: Klin. Monatsbl. f. Augenh. **50**:159, 1912.

4. van der Hoeve, J.: Nervenfaserdefekte bei Retinochoroiditis juxtapapillaris, Klin. Monatsbl. f. Augenh. **18**:487, 1914.

5. Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 5, p. 546.

6. Abraham, S. V.: Retinitis Juxtapapillaris—Jensen, Arch. Ophth. **2**:452 (Oct.) 1929; Chorioretinitis Juxtapapillaris (Jensen), ibid. **8**:503 (Oct.) 1932.

7. Dimmer: Chorioretinitis juxtapapillaris, Der Augenspiegel, ed. 3, Vienna, Franz Deuticke, 1921, p. 577.

CONCLUSION

The following facts are significant in the case presented: the sudden onset of the disease in a young and apparently healthy adult, the tremendous optic neuritis that veils the true picture of the disease, the location of the lesion in the anterior layers of the retina at the margin of the disk with no involvement of the choroid or the pigment epithelium, the presence of the correspondingly constant sector defect of the field, which includes the blind spot and extends to the periphery, and the negative Mantoux test and test with new tuberculin, plus the absence of signs in the roentgenogram of the chest of an old or a new tuberculous process in a disease that is considered by many as tuberculous.

STEREOSCOPIC EXERCISES IN AMETROPIA

A NEW USE OF THE STEREOSCOPE FOR DEVELOPING TOLERANCE TO THE LENSES CORRECTING ERRORS OF REFRACTION

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Every one is familiar with the use of the stereoscope for developing binocular vision, fusion amplitude, stereopsis and duction power. It is my purpose in this discussion to present an additional use of the stereoscope unconnected with any of the aforementioned uses. To understand the bearing of this application of the stereoscope, it is necessary to consider a brief preliminary discussion on some of the causes of discomfort incident to the first wearing of correcting lenses.

Of the several causes responsible, unquestionably one important cause is the fact that the lenses tend to break up old established ocular habits. The person with hyperopia who is given his first plus correction had for a time previous to his correction established ocular habits which served his needs even though under strain. In uncomplicated cases, he had developed a habit of forcing his accommodation ahead of his convergence. If the hyperopia was 2.00 diopters, for example, the subject had to learn to accommodate 2.00 diopters more than normal and at the same time learn to restrain his convergence from following suit. In the course of time he learned to stimulate his accommodation excessively, but his convergence, only mildly; i. e., a relatively small convergence effort became habitually associated with a relatively high accommodative effort.

Now the plus lenses which correct the hyperopia create new needs. The patient is forced to inhibit his accommodation to the extent of the plus power in the correcting lens. And, relative to the accommodation now used, he has to increase his convergence stimulus. As an example, before correction he had to accommodate 2.00 diopters for clear, distant vision and suppress all convergence. He thus gradually acquired the habit of accommodating 2.00 diopters without stimulating the convergence. But with the correction, he has to learn to stimulate his convergence 2 meter angles when he accommodates 2.00 diopters. In short, he has to acquire the new habit of stimulating his accommodation less and his convergence more.

A person with uncorrected myopia has likewise acquired unbalanced ocular habits, though of a different nature. He had to learn to lessen

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or suppress accommodative stimuli while increasing convergence stimuli. For example, a person with a myopia of 1.00 diopter, when looking at an object 1 meter away, had to learn to suppress all accommodation while exercising a convergence of 1 meter angle. With the correction on, the patient has to unlearn the old habits of making a great convergence effort to a slight accommodative effort and learn to make full accommodative effort to a relatively small convergence effort. As another instance, whereas before correction the person with myopia of 1.00 diopter fixing at 50 cm. had learned to converge 2 meter angles to an accommodation of 1.00 diopter, he now has to learn to converge but 1 meter angle to an accommodation of 1.00 diopter. In brief, he has to unlearn the habit of suppressing accommodation and stimulating convergence and learn the new habit of stimulating accommodation and relatively suppressing convergence.

This process of learning to establish a new relationship between the accommodation and convergence is acquired easily by some and rather painfully by others. This is one reason why some people find their glasses comfortable from the beginning, while others have to pass through a more or less trying period. The stereoscope offers a means of facilitating the change of ocular habits by means of a few simple stereoscopic exercises.

It is the aim of the correcting lenses to establish a normal relationship between the accommodation and convergence primarily so that the efforts for the two activities will be equal and balanced. As a general principle, where two associated activities are unbalanced in that one outruns the other, in order to obtain balance or equilibrium it is desirable to reverse the order and for a time make the less active function the more active. That is, one must overdo the correction, so to say, swing the pendulum far in the opposite direction so that by swinging back, equilibrium will be established. This can be done with the stereoscope.

The ordinary stereoscope has a plus 5.25 diopter lens and an 8 diopter prism base out before each eye. The distance between the centers of the lenses is about 60 mm., corresponding to the average pupillary distance. The rod of the stereoscope provides for movement of the chart from about 9 to 23 cm. The focus of the lenses is about 19 cm. and represents the furthest distance a person with emmetropia could possibly see anything clearly. By bringing the chart as near as possible, say, to 9 cm., the accommodation exerted is 5.75 diopters (11.00 diopters less 5.25 diopters). Thus, if a stereogram carrying small type is moved from 19 to 9 cm., the accommodation required to keep the type clearly in focus will increase from zero at 19 cm. to 5.75 diopters at 9 cm., and decrease from this amount to zero again as the chart is withdrawn to 19 cm. By moving the stereogram back and

forth on the rod of the stereoscope, one can produce a gradual increase or decrease of accommodation from zero to 5.75 diopters.

But with this change of accommodation, there is inevitably associated a change in convergence, the extent of which depends partly on the distance between the two halves of the stereogram. The displacement produced by the prisms will be 16 per cent of the distance of the chart, as each prism diopter causes a displacement of 1 per cent of the distance. For convenience, if 10 cm. is taken as the nearest point at which the chart will be brought, the displacement at that distance will be 16 per cent of 100 mm., or 16 mm. The displacement will increase 16 per cent of 1 cm., i. e., 1.6 mm. for every centimeter that the chart is placed beyond 10 cm.

If the distance between the two halves of the stereogram is made 76 mm., the displacement of 16 mm. at 10 cm. will bring the images of the corresponding parts of the stereogram to 60 mm. apart. As this corresponds to the average pupillary distance, the visual axes in blending the images at 10 cm. are parallel. The convergence then with chart at 10 cm. is zero. As the chart is moved further away, the displacement increases, and the distance between the corresponding parts of the stereogram becomes successively less. This will necessitate increased convergence of the visual axes in order to blend the images. At the limit of 19 cm., the convergence required will be about 8 prism diopters. It will be seen that as the chart is moved away from the eyes, more convergence is called for, reaching its maximum at 19 cm. As the chart is moved nearer to the eyes, less convergence is called into play, reaching its minimum, which is zero, at 10 cm.

It will now be seen that the stereoscope provides a mechanism which in its effect on the associated accommodation and convergence is unlike any other mechanism. It is unique in that it affects the two functions in a reciprocal way. Moving the stereogram so as to increase convergence activity causes a corresponding decrease of accommodative activity. The two functions can therefore be made to work in an unusual relationship, specifically in a way contrary to the normal relationship, where increase or decrease of one function is associated with a corresponding increase or decrease of the other function. In the stereoscope, moreover, these reciprocal changes are gradual and continuous during the movement of the chart.

The accompanying table will show the amounts of accommodation and convergence produced by a stereogram of 76 mm. separation between its corresponding parts at different points (intervals of 1 cm.) between the limits of 10 and 19 cm. The accommodation is taken to the nearest quarter diopter and the prism calculated to one decimal point. Minor discrepancies such as result from the distance of the eyes from the stereoscopic lenses and oblique incidence have been ignored.

It may be of interest to show briefly how the table is obtained. The accommodation exerted at each point is simply the amount corresponding to that distance less the plus 5.25 diopters in the stereoscope. For example, at 16 cm. the total power required is 6.25 diopters (16 into 100); this less 5.25 gives 1.00 diopter. The amount of convergence exerted is easily obtained by noting the displacement beyond 60 (60 mm. corresponding to parallelism of the visual axes) and determining the prism value of such displacement. For example, at 10 cm. the convergence is zero because the two 8 diopter prisms cause a displacement of 16 mm., thereby placing the corresponding images of the objects which are 76 mm. apart only 60 mm. apart, and thus adapting the images to parallel visual axes. At 11 cm. there is an increase of displacement of 1.6 mm. (16 per cent of 1 cm.). To find the prism value of 1.6 mm. displacement at 11 cm., or 110 mm., one divides

Accommodation and Convergence

Distance, Cm.	Accommodation, Diopters	Convergence, Prism Diopters
10	4.75	Zero
11	3.75	1.5
12	3.00	2.7
13	2.50	3.7
14	2.00	4.6
15	1.50	5.3
16	1.00	6.0
17	0.75	6.6
18	0.25	7.1
19	Zero	7.6

1.6 by 110 to three decimal places, and multiplies the result by 100 to get prism diopters to one decimal place. It is identical with the process of finding what per cent 1.6 is of 110. Dividing 1.6 by 110 gives 0.015, and therefore by multiplying by 100, 1.5 prism diopters. For example, at 16 cm. the displacement beyond 60 is 6×1.6, which equals 9.6. Dividing this by 160 and multiplying by 100 gives 6 prism diopters.

In hyperopia the habit to be broken is the one which associates excessive accommodative effort with repressed convergence effort. By placing the stereogram at 10 cm. and having the patient read the words or pick out single letters and continue to do so while the chart is gradually moved further off, one forces a decrease of accommodative effort with an increase of convergence effort. This tendency is undesirable as a final stage, but is merely brought into use as an exercise to help get equilibrium. In myopia, the habit to be broken is the one which associated repressed accommodative effort with excessive convergence effort. By placing the stereogram at 19 cm., or a little nearer if the patient cannot relax enough to see the chart, and having the patient read the words or pick out single letters and continue to do so while the chart is gradually brought nearer, one forces an increase of

accommodative effort with a decrease of convergence effort. This is, of course, just the reverse of the way the two functions were working previous to the correction, and several exercises of this sort will facilitate the breaking of the old habit in favor of one establishing equilibrium.

A stereogram for this work can easily be made by pasting two strips of identical small type, say, 2 cm. wide by 5 cm. long from duplicate newspapers or magazines on a piece of cardboard 7.5 by 17 cm. to correspond in size to the regular stereoscopic cards. The strips are to be pasted on so that their centers are 76 mm. apart. A control mark such as a heavy dot placed above the right strip and below the left strip must be added in order to make sure of the binocular reading of the chart. Wider strips, e. g., a column from the newspaper, may also be used. These, however, suffer at the lateral margins from slightly different deviations in the two prisms, because the corresponding parts at the margins are affected by "apex incidence" in one prism and "base incidence" in the other. But these slight aberrations are of little consequence and are in fact found to a slight extent even with the narrow strips.

SUMMARY AND CONCLUSION

The discomfort incident to the first wearing of lenses for ametropia is largely due to the necessity of breaking up old ocular habits in favor of new ones. The habitual adjustment between the accommodation and convergence before correction was either excessive stimulation of the accommodation with slight stimulation of the convergence in hyperopia, or excessive stimulation of the convergence with slight stimulation of the accommodation in myopia. The correcting lenses aim to bring about a condition of equal and balanced stimulation between the accommodation and convergence. To attain this balance more readily, it is desirable as an exercise to reverse the habitually faulty accommodation-convergence relationship in a manner contrary to the old habit. Exercises which overcorrect the faulty association, so to say, swing the pendulum too much in the opposite direction, which by recoil tends to get into position of equilibrium. The stereoscope enables one to conduct such exercises.

PARTIAL CORTICAL BLINDNESS WITH PRESERVATION OF COLOR VISION

REPORT OF A CASE FOLLOWING ASPHYXIA (CARBON MONOXIDE POISONING?); A CONSIDERATION OF THE QUESTION OF COLOR VISION AND ITS CORTICAL LOCALIZATION

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It is a fact long established by clinical observation and pathologic studies that cortical and subcortical lesions may give rise to dissociation of color perception from visual acuity, namely, loss of the former without impairment of the latter; and it is well known that in impairment of vision, whether peripheral or central, the first to be lost and the last to be regained is the perception of color. In addition to this dissociation between color and light perception, other types of dissociation in the recognition and naming of various colors have been described. Paralleling the disturbances in the realms of speech and general sensation, distinction is made between color agnosia and loss of color vision. For instance, a patient may fail to recognize or distinguish colors and yet retain the memory of color. These dissociations and the pathologic and psychologic problems to which they give rise, and particularly the question of cerebral localization, have been the subject of rather extensive, though inconclusive, studies. The question of localization has been particularly perplexing, not only as to what part of the cortex is affected in the case of color blindness, but as to whether the lesion causing such loss is cortical or subcortical. This leaves out of consideration the color receptors in the retina and the primary visual centers (lateral geniculates). Recently a case has come under my observation which justifies raising the question once more and, despite the absence of pathologic studies, permits theoretical speculation based on clinical observation.

While the literature is full of reports of cases in which there was achromatopsia or hemichromatopsia in the presence of good acuity of vision, the reverse, namely, impairment of visual acuity and the preservation of color perception, has, as far as can be ascertained, not been described. The present report deals with such an instance, and is

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Read at the International Neurological Congress, Bern, Switzerland, 1931.
From the Neurological Department of the Montefiore Hospital, New York.

thought worthy of record because of its uniqueness. Before discussing the question of color localization and its psychic elaboration and a few other implications, it may be well first to give a detailed description of the case.

REPORT OF CASE

H. B., a boy of 13 years and 7 months was admitted to the Montefiore Hospital in August, 1930, with blindness and mental deterioration as the chief complaints. The history was that, with the exception of pneumonia as an infant and measles at a later age, the boy had been perfectly well up to one year before admission. Mentally he had been rather advanced for his age, having entered the first year of high school at 12 years. On Aug. 29, 1929, he was overcome by the smoke of a fire which broke out in his home, in which five members of his family perished and he, together with his father and one sister, survived. All three were unconscious, the boy for about two hours, and were resuscitated by means of pulmotors. When the boy revived at Bellevue Hospital, it was found that he was totally blind and that he could neither hear nor speak. The records state that the pupils were equal and regular and reacted to light. Both fundi showed recent hemorrhages. He had rhythmic twitchings of the left side of the face, there was a left-sided Babinski sign and both hands were in flexion. Within one week he began to hear and to speak, how distinctly it is not stated. He could walk after two months, but he remained totally blind. During his five months' stay in the hospital he had occasional convulsive seizures with decerebrate rigidity attitudes. In March, 1930, he was observed at another neurologic hospital where it was noted that he had begun to perceive light.

On admission to the Montefiore Hospital, neurologic examination showed the following: The left upper extremity was held in flexion at the elbow and wrist, and there was bilateral loss of associated movements in the arms. The patient had some difficulty in the performance of skilled acts, which was due in part to weakness and in part to apraxia. He showed associated movements (reflexes) in one arm on gripping the examiner's hand with the other. He had no incoordination and no abnormal involuntary movements. All the deep reflexes were hyperactive, the abdominal reflexes were present, and the Babinski toe sign was positive on both sides. The left upper extremity was hypertonic; the other limbs showed normal tonus, while the fingers and toes were hypotonic. There were bilateral pes cavus and some equinovarus. Pain, touch, temperature and vibration were perceived normally throughout, but there was impairment of the position sense, of two point discrimination and of the stereognostic sense in the fingers of both hands.

Smell was intact. The pupillary reflexes were normally active, the fundi were normal and there were no ocular palsies, but there was rotatory nystagmus (which, from the history, was definitely congenital). Visual examination showed the following: Form perception was markedly disturbed. The patient could not recognize small objects, such as a coin, key, pen or watch, and large objects, such as a book, newspaper or telephone, he could distinguish with difficulty. Color perception, on the other hand, was quick and accurate. He recognized not only all primary colors, but such shades as brown and pink. He knew at once the colors of small objects which he could neither name nor tell the form of. He picked out colors on command. Central vision for colors seemed to be better than peripheral. While it was impossible to outline the fields of vision, it seemed by the way he held his head in gazing at an object that there was some degree of

hemianopic defect, and that he used his lower fields in fixation. He groped in trying to grasp objects, and sometimes collided with persons and things, apparently because he could not estimate distance and probably as a result of a defect in visual spatial discrimination. There was right supranuclear facial weakness; hearing was normal; the tongue was protruded in the midline; the rest of the cerebral nerves were intact. Speech was unintelligible and dysarthric, but there was no definite evidence either of motor or of sensory aphasia. The patient could write, though he showed some apraxia in the execution of skilled acts, which was, however, difficult to evaluate because of the astereognosis. He showed intellectual deterioration, emotional inadequacy and lack of insight. His mental age, on psychometric examination, was given as 7 years and 8 months and his intelligence quotient as 52. All the laboratory tests yielded negative results, while the general physical condition of the patient was good. During the year following his admission to the hospital his mental condition improved but slightly and his vision returned somewhat, in that he could walk through the wards without colliding.

ANALYSIS OF THE CASE

From the early records of the case and the more recent physical findings it would seem as if the boy suffered a brain lesion in which the cortex was extensively affected, while the subcortical and basilar structures were involved to a lesser extent. The prolonged period of unconsciousness no doubt points to cortical involvement and so possibly do the early convulsions, although the decerebrate postures mentioned point to lower levels. The loss of hearing might be attributed to temporary bilateral impairment of the temporal lobe, but the loss of speech was not entirely due to cortical involvement. The loss of vision, aside from the early temporary retinal hemorrhages, was recognized as cortical because of the preservation of pupillary reflexes and subsequent absence of optic atrophy, not to mention the disturbances in spatial discrimination, the visuopsychic defect and the subsequent relative dissociation in color and light vision. The probable bilateral hemianopia and the defect in the upper fields can also be correlated with occipital, that is, calcarine disease. Further evidence of parietal cortical involvement may be deduced from the type of sensory loss, namely, position sense, point discrimination and stereognosis, and possibly the hypotonia of the fingers. The bilateral pyramidal tract signs (the Babinski phenomena) may possibly be attributed to a central cortical lesion. Finally, impairment of both frontal cortices may be inferred from the grave mental deterioration, the boy having descended from a superior mental level to very defective mentality. The loss of associated movements, however, was suggestive of basal ganglion origin, and so were possibly the attitude and posture of the feet.

It is of interest that other nerve structures, either peripheral or central, were apparently spared. This raises the question of the kind of noxious agent which brought about the clinical picture. The first assumption was that of carbon monoxide poisoning, but it is known that

smoke contains, in addition to carbon monoxide, carbon dioxide, methane, carbon particles, sulphur and ammonia gases and possibly many others. But carbon monoxide alone could well induce the lesion assumed to have existed in the patient. It is true that carbon monoxide has a predilection for the basal ganglions, especially causing lenticular softening, and for the peripheral nerves, but no part of the nervous system is immune to the poison. That parkinsonian syndromes and peripheral nerve palsies frequently characterize carbon monoxide poisoning is true, but the mental deterioration and psychotic manifestations so commonly encountered attest the additional cortical involvement.

REVIEW OF THE LITERATURE

Without entering into a description of the cerebral pathology of gas poisoning, reference may be made to the works of Hill and Semack,[1] Stewart [2] and Swetnik.[3] A review of the literature of carbon monoxide poisoning, with special reference to involvement of the cortex causing mental impairment, but more particularly cortical or subcortical loss of vision and unilateral or bilateral hemianopia, fails to show an instance of preservation of color vision in the presence of impairment of acuity of vision. Always the reverse was true. Uhthoff [4] does not mention the subject of color blindness in carbon monoxide poisoning. One of the earliest references is given by Bourdon.[5] Sibelius [6] reported a case of permanent cortical blindness, the result of softening of both occipital lobes. Abt and Witt [7] described the case of a boy asphyxiated by gas, who had loss of vision and preservation of the pupillary reflexes. Friedenwald reported a case of hemianopia following poisoning by illuminating gas. Abelsdorff's [8] patient began to lose vision four days after carbon monoxide poisoning. The fundi were normal, the pupils reacted to light and in accommodation, visual

1. Hill and Semack: Changes in the Brain in Gas (CO) Poisoning, Tr. Chicago Path. Soc. **10**:263 (April) 1918.

2. Stewart, R. M.: A Contribution to the Histopathology of Carbon-Monoxide Poisoning, J. Neurol. & Psychopath. **1**:105, 1920.

3. Swetnik, Z.: Monatschr. f. Psychiat. u. Neurol. **94**:71 (Nov.) 1929.

4. Uhthoff: Die Augenveränderungen bei Vergiftungen, in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1911, vol. 11, pt. 2a, p. 124.

5. Bourdon, H.: Des paralysies consécutives à l'asphyxie par la vapeur du charbon, Thèse de Paris, 1843.

6. Sibelius: Die psychischen Störungen nach akuter Kohlenoxydvergiftung, Monatschr. f. Psychiat. **18**:39, 1905.

7. Abt, I., and Witt, D. B.: Cortical Blindness and Mental Deterioration in a Child Due to Carbon Monoxid, M. Clin. North America **5**:1645 (May) 1922.

8. Abelsdorff: Vorübergehende Erblindung mit Augenmuskellähmung nach Kohlenoxydvergiftung, Deutsche med. Wchnschr. **46**:210, 1920.

acuity was impaired, but color perception was entirely lost. The patient gradually recovered. Fejer [9] described the case of a man of 60 who, following asphyxia by illuminating gas, became blind, though his pupils and fundi were normal; that is, he had cortical blindness. He gradually recovered vision within three weeks, and by the end of the fourth week began to have color perception.

While cases of achromatopsia and hemichromatopsia are comparatively uncommon, a number due to various causes have been reported. Brill [10] described color blindness following apoplexy. At autopsy he found softening of the inner surface of the occipital lobe, the cortex bordering on the calcarine fissure, the upper edge of the lingual gyrus and the lower half of the cuneus. On the other hand, MacKay and Dunlop [11] reported the case of an old man who at first had partial double hemianopia, which gradually receded and left behind complete and permanent color blindness. At autopsy they found lesions in both occipital lobes, in the occipito-temporal and especially in the fusiform lobes. They stress the fact that the calcarine area was spared and that the white fibers entering it were affected. They cited Verry's case in which hemichromatopsia was caused by a lesion of the white matter of the lingual and fusiform convolutions and the posterior inferior portion of the cuneus. Here also it was pointed out that the cortex was spared. MacKay and Dunlop concluded that color is perceived in the posterior parts of the lingual and fusiform convolutions. Swanzey [12] reported a case of hemichromatopsia, without changes in the fundus, probably cortical in location and apoplectic in origin. Lenz [13] reported a case of pure hemichromatopsia which at autopsy showed atrophy of the calcarine cortex, principally of the superior pyramidal layers. He also described the brain of a rabbit which lived in a blue light for thirteen months (from birth), in which he found rarefaction of the same layers. Several years later he [14] described another case in which there was "disturbance of color perception." There he found marked involvement of the white substance bilaterally, especially the gyrus fusiformis. The calcarine cortex was less involved. Wilbrandt [15]

9. Fejer, Gyula: Cortical Blindness Due to Carbon Monoxid, Am. J. Ophth. **7**:522 (July) 1924.

10. Brill, N. E.: Am. J. Neurol. & Psychiat. **1**:356, 1882.

11. MacKay, G., and Dunlop, J. C.: Scottish M. & S. J. **5**:503, 1899.

12. Swanzey, H. R.: Tr. Ophth. Soc. U. Kingdom **3**:185 (July 6) 1883.

13. Lenz, G.: Zur Pathologie der cerebralen Sehbahn unter besonderer Berücksichtungen ihrer Ergebnisse für die Anatomie und Physiologie, Arch. f. Ophth. **72**:1 and 197 (Oct.) 1909.

14. Lenz, G.: Zwei Sektionsfälle doppelseitiger zentrale Farbenhemianopsie, Ztschr. f. d. ges. Neurol. u. Psychiat. **71**:135, 1921.

15. Wilbrandt, H.: Ophthalmiatrische Beiträge zur Diagnostik der Gehirnkrankheiten, Wiesbaden, J. F. Bergmann, 1884.

believed that color perception occurred in the inferior portion of the visual cortex and the inferior portion of the optic radiation. He was further of the opinion that the occurrence of hemichromatopsia in superficial lesions must be attributed to interference with fibers running between the gyrus angularis and the inferior portion of the visual area.

Samuelsohn [16] described a case of hemichromatopsia caused by glioma of the optic tract, thalamus and geniculate body, which spared the cortex. He quoted a case reported by Steffan in which a printer, following an apoplectic attack of dizziness, lost color perception, retained light vision and had normal eyegrounds. Halpern and Hoff,[17] in discussing cerebral color blindness, make a distinction, as others do, between color sense and color naming, and in agreement with Pötzl and Lenz localize the former in cell groups of the second and third layers of the area striata. They also mention loss of color vision in lesions of the angular gyrus and of the junction of the temporal and occipital lobes. Gelb reported two patients who lost the ability to see "surface colors," while Poppelreuter spoke of an absolute color sense. Best [18] thought that there was a "color field" in the left hemisphere. Marie, on the other hand, saw no reason for postulating a special center for color. A point worth mentioning, which points to cortical color representation, is the occurrence of fortification scotoma in which the colors of the spectrum appear as a migrainous aura. Lenz believed that there was no color perception outside the visual area.

Klüver [19] has given a very complete résumé of the literature on visual disturbances after cerebral lesions, but the best study is undoubtedly that of Pötzl.[20] He concludes that patients with word blindness also have disturbances in color vision; that in psychic blindness there is color agnosia, but that there may be optic agnosia without loss of ability to name colors. Pötzl quotes Gelb and Goldstein who also speak of color-naming aphasia; color aphasia they regard as part of optic aphasia. Pötzl stresses the possibility of selective color loss, e. g., of blue, green, yellow, red, etc. As to localization, he thinks that the color sense resides either in the calcarine area or in its projection system, but that color agnosia is the result of a lesion in adjacent

16. Samuelsohn: Ein Fall von Hemichromatopsie, Berl. klin. Wchnschr. **26**: 331, 1890.

17. Halpern, F., and Hoff, H.: Kasuistische Beiträge zur Frage der cerebralen Farbenblindheit, Ztschr. f. d. ges. Neurol. u. Psychiat. **122**:575, 1929.

18. Best, F.: Zentralbl. f. d. ges. Ophth. **3**:193 and 241, 1920.

19. Klüver, H.: Visual Disturbances After Cerebral Lesions, Psychol. Bull. **24**:316 (June) 1927 (review of the literature).

20. Pötzl, Otto: Die optisch-agnostischen Störungen, in Aschaffenburg, G.: Handbuch der Psychiatrie, Vienna, Franz Deuticke, 1928, vol. 1, p. 95.

regions; also that the oral and basal portions of the visual pathways pertain to color perception. Since in recovery of color vision the perception of certain colors is regained first, he concludes that there are specific groups of color-perceiving cells lying next to one another. Isakower and Schilder [21] reported a case with optic agnostic disturbances following carbon monoxide poisoning. Pohlisch [22] also cited cases of amaurosis and loss of color vision in coal gas poisoning. Mills [23] was of the opinion that color was perceived in the lingual and fusiform lobes, but that blending occurred both peripherally (retina) and cortically. He thought that only fundamental colors had cortical representation; that hue, which is based on wave length, tint, on the amount of white light present, and brightness, on the amount of black mixed with color (inverse ratio), depended on the retina. Henschen [24] was of the opinion that there were special cells for color in the calcarine area and the geniculate ganglions. He had examined night monkeys which showed light but no color cells, whereas day monkeys had both. In the night monkeys he found only one type of ganglion cells in the fourth layer, whereas the day monkeys had a second layer, which he designated as color cells. It will be recalled that Henschen held to the view that there is exact localization in the cortex of the various portions of the retina, the macula also having specific localization. Monakow, on the other hand, contended that the fibers were scattered throughout the geniculate, optic radiation and occipital cortex. Brouwer,[25] compromising, as it were, admitted that the fibers are widely distributed in the geniculate bodies, but that there is some degree of localization; also that while the fibers do go to the occipital poles, they are not exclusively localized there.

In a review of two hundred cases of cortical blindness Magitot and Hartmann [26] stressed the early loss of color vision, followed by the loss

21. Isakower and Schilder: Ztschr. f. d. ges. Neurol. u. Psychiat. **113**:102, 1928.

22. Pohlisch, K.: Das psychiatrisch-neurologische Bild der Kohlenoxydvergiftung, Monatschr. f. Psychiat. u. Neurol. **70**:339, 1928; **71**:82, 1929 (literature).

23. Mills, C. K., in Posey and Spiller: The Eye and the Nervous System, Philadelphia, J. B. Lippincott Company, 1906, p. 88.

24. Henschen, S. E.: Lichtsinnzellen und Farbensinnzellen im Hirn, Hygiea **91**:705, 1929; **92**:585, 1930; Ztschr. f. d. ges. Neurol. u. Psychiat. **55**:677, 1930; **59**:164, 1931. Henschen, S. E.: Klinische und anatomische Beiträge zur Pathologie des Gehirns, published by the author, 1930; abstr., Klin. Wchnschr. **9**:1600, 1930.

25. Brouwer, B., and Zeeman, W. P. C.: Projection of the Retina in the Primary Optic Neurons in Monkeys, Brain **49**:1 (March) 1926.

26. Magitot, A., and Hartmann, E.: La cécité corticale, Bull. Soc. d'opht. de Paris, 1926, pp. 427 and 639.

of perception of movement and finally by the perception of light vision; also that recovery takes place in the inverse order. They pointed out that in pure cortical blindness from surface occipital lesions the patient has memory for visual images which lack color. Lereboullet and Mouzon [27] cited such a case. Holmes and Horrax [28] described a case with loss of spatial and topographic memory and of stereoscopic and color vision, yet the patient had memory of colors. The loss of color vision was greater than that of light perception. He also had bilateral inferior hemianopia. The lesion, due to gunshot, was said to have been in both angular gyri.

There are a great many references in the literature to the mental changes in patients who survived gas poisoning. These range from simple memory defect to complete dementia. Mayer [29] mentioned among other manifestations, amnestic disturbances. Stengel [30] spoke of manic-depressive and schizophrenic syndromes. Pohlisch [22] also recorded, in addition to amnesias and paraphasias, psychotic syndromes of the manic-depressive and schizophrenic varieties and others characterized by paranoid, confused or catatonic states.

COMMENT

It is generally hazardous and always of doubtful validity to draw conclusions as to anatomic localization from clinical observations unsupported by pathologic studies. But the uniqueness of the case herein reported and the richness of the literature bearing on the subject of color vision permit a few inferences. From the analysis of the case the assumption is justified that the lesion was predominantly though not exclusively cortical. Bearing in mind the cortical type of blindness (occipital), the early loss of hearing (bilateral temporal), the cortical type of sensory loss (parietal), the mental deterioration (frontal lobes) and the early convulsions, it would seem as if the whole cortex had been affected.

The structural as well as functional "decortication" may well have been due to involvement of the surface layers of the brain by the pathologic process. The pial engorgement so commonly observed in gas poisoning, and the minute hemorrhages, may have been just sufficient to affect the cortex and to spare, in the main, the subcortical structures. But whatever the pathologic substratum was, clinically

27. Lereboullet and Mouzon: Paris méd. **2**:19, 1917.

28. Holmes, G., and Horrax, G.: Spatial Orientation and Visual Attention with Loss of Stereoscopic Vision, Arch. Neurol. & Psychiat. **1**:389, 1919.

29. Mayer, A.: Klinisch-anatomische Erfahrungen bei Kohlenoxydvergiftung des Zentralnervensystems, Klin. Wchnschr. **6**:145 (Jan. 22) 1927.

30. Stengel, E.: Zur Kenntnis psychischer Erkrankungen nach Leuchtgasvergiftung, Ztschr. f. d. ges. Neurol. u. Psychiat. **122**:587, 1929.

there is no doubt that the cortex bore the brunt of the damage. The impairment of vision and preservation of color perception point to cortical involvement, and fit in with most of the observations recorded in the literature. It is worth emphasizing that the majority of investigators previously cited concur in the view that color perception probably takes place in the third or fourth layers of the calcarine region, but that memory for colors is a higher psychic elaboration, which takes place on the outer surface of the occipital lobe adjoining the temporal convolutions and the angular gyrus. The formed visual hallucinations, frequently colored, occurring in hemianopic fields, which are generally attributed to involvement of the occipitotemporal regions, and the migrainous fortification scotoma lend additional evidence.

Whatever the anatomico-pathologic explanation is, psychologically the case may be interpreted from the point of view of a disturbance in symbolic thinking. The patient's vision was impaired: He saw objects without being able accurately to recognize or properly to name them, especially if they were small. His greatest difficulty, then, was in the sphere of higher psychic elaboration, in the realm of symbolic thinking, and one may conclude that he suffered from mind or psychic blindness and not from disturbance in primary visual perception. It will be recalled that when shown a coin he knew that it was an object but could not tell what kind of an object it was. This obviously is an aphasic disturbance, and yet the patient understood speech and carried out commands well. That he had memory for colors and retained the power of recognizing and naming colors, even to the point of distinguishing between various shades, shows that just as there can be color agnosia without psychic blindness so may there be psychic blindness without color agnosia. This contradicts Pötzl's view that all cases of psychic blindness show color agnosia. The interest of the case, therefore, lies in the unusual type of dissociation.

There remains one question: If color vision resides in the cortex and the surface of the brain was destroyed by the pathologic process, color vision should have been lost and not retained. The most probable answer is that either certain parts of the cortex or the layers presumed to perceive and elaborate color vision escaped destruction or they recovered while the visual areas did not. It is barely possible that there are special color-conducting fibers, but all evidence at hand points to the cortex as the seat of color vision. In any event, the case herein presented warrants the statement that color perception and visual acuity can be dissociated in such a way that the former is preserved while the latter is impaired.

CATARACT AND DIABETES

STUDY I

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The field of the relation of disturbances of carbohydrate metabolism to cataract was chosen as one fertile for research into the cause of cataract because of the well known frequency of changes in refraction and development of lens opacities in diabetic subjects. Berndt as early as 1834 reported diabetes as a cause of cataract. Duke-Elder differentiated two types of cataract occurring in diabetes: (1) the form of senile cataract appearing frequently and at a relatively early age and (2) a characteristic form peculiar to the disease occurring more rarely. Duke-Elder felt that the diabetic state, by subjecting the lens to an abnormal traffic of fluid, induced the condition and tended to alter its type. He thought that the denaturing action of radiant energy in the presence of excessive sugar and acetone was probably the main factor in the production of cataract in diabetic persons. One must also consider the general dehydration of blood and tissues and the decrease in the permeability of tissues and membranes. This affects the semipermeable barrier through which the aqueous humor is dialyzed, the lens capsule and the membranes of the lens epithelial cells and the lens fibers.

The abnormal traffic of fluid to which the lens is subjected is produced by the changes in the osmotic pressure of the blood, tissues, tissue fluids and humors due to variations in the sugar content. As the blood sugar rises, the salt content must necessarily be lowered to maintain the osmotic balance. The larger sugar molecule diffuses through the barrier of tissue between the capillary blood in the ciliary processes and the anterior chamber more slowly than does the smaller salt molecule. Hence, the aqueous formed in the presence of increased blood sugar probably has a lesser osmotic pressure. As the lens substance is hygroscopic, it has an affinity for water, and as it is bathed by a fluid of lowered osmotic pressure, it tends to take up additional water.

The investigations were conducted under the auspices of the American Academy of Ophthalmology and Oto-Laryngology, of which I was research Fellow, and of the Department of Ophthalmology of the College of Physicians and Surgeons of Columbia University.

For references, tables, figures and experimental data, the reader is referred to the monograph "A Study of Disturbances of Carbohydrate Metabolism in Relation to Cataract" published as a supplement to the Transactions of the American Academy of Ophthalmology and Oto-Laryngology, 1932.

If the process of hydration is relatively mild, the lens swells and is deformed, its curvature and refractive power are increased, and the eye tends toward myopia. The site of the change in refraction is most probably in the lens, the capsule and substance of which are freely permeable to sugar. The theories concerning the effect on the ciliary muscle or concerning the change in the index of refraction are less tenable. The effect of fluid of lowered osmotic tension in producing changes in refraction is not constant in all diabetic patients or even in the same one. The recovery from the conditions of increased blood sugar produces a relative hyperopia by reducing or removing the myopia of an increased sugar content or an absolute change in producing hyperopia greater than the average focus for the patient previously. Such changes are seen more frequently now with the increased use of insulin than formerly through the effect of dietary restriction.

If the process of hydration of the lens is more severe and the inflow of fluid more rapid, droplets are formed under the capsule, and the transparency of the lens is impaired by the irregular reflection of light at the interfaces between the relatively pliable cortex and the hard, resistant lens nucleus. Vacuoles or globules have been noted frequently in the lenses of diabetic patients, and the water-splitting of the sutures and the separation of the lamellae are characteristic of the early form of senile cortical cataract occurring in elderly diabetic patients. The water which effects this is in fact a colloidal solution. These changes do not occur uniformly in all diabetic patients, nor are the phenomena consistent even in the same person. It appears almost impossible to explain the factors on a purely physicochemical basis, and much as one may dislike the idea, one must assume a vital control process.

The rare form of rapidly developing cataract in diabetic patients, which occurs usually in the younger patients, develops in the presence of a high concentration of sugar and usually when the patient is fairly well, i. e., when he has a sufficient reserve of fluid. It is not associated with the state of acidosis or coma, although it may occur in those who have passed through these episodes. It is generally bilateral and starts just beneath the capsules. It occurs in very young patients; in three cases the patients were 11 months, 5 years and 7 years of age, respectively. The cataract matures rapidly; in one case the process took four days and in another, eleven days. In a case which my associates and I observed in an adolescent of 16 years, the cataract reached the immature stage in four weeks, the vision being reduced to 3/200. An idea of the rarity of the condition may be gained from the fact that this was the only typical case which we observed over a period of eight years, even though we were looking for such cases. Weeks saw but two, perhaps three, in his whole experience of forty-five years of practice.

In our case there were anterior and posterior subcapsular opacities made up of minute globules, which were more numerous and confluent

at the posterior pole. The cortex was clouded with punctate and flaky particles, and the lens sutures were separated by large spindle-shaped lacunae.

Fischer reported a case in a girl of 17 years in whom the condition cleared after prolonged treatment with insulin. A few similar experiences have been recorded. It is possible that the early changes which do not lead to denaturation of the lens protein may be cleared up, but after denaturation and coagulation have occurred, it is to be doubted that a reversal of the process is possible. However, proper treatment may arrest the process and effect an improvement.

Some investigators, including Fuchs, have felt that the opposite process of dehydration is at work to produce the cataract of diabetes as it does in cholera by the abstraction of water from the lens, but here again Duke-Elder contends that the loss of the dialyzable and osmotically active salt constituents of the blood, while making the blood more viscous, will in the osmotic sense render it more dilute and thus produce the conditions that I referred to before. The incidence of hydropic swelling of the pigment epithelium of the iris and the ciliary body in sections of eyes of diabetic patients is familiar, as is also the separation of the layers of pigment and the dispersion of pigment at the time of the iridectomy in diabetic patients with cataract.

Disturbances of fat metabolism are common in diabetes, and fatty substances are found in increased amounts in cataractous lenses. Friedenwald said that there is always a tendency to incriminate an altered fat metabolism, but that at present there is no evidence to warrant this. However, it is possible that the lipoids which normally exist in the lens cells and fibers and in the intercellular and interfibrillar spaces in a state of fine emulsion and balance are disturbed by certain metabolic reactions, such as the chemical process of aging or because of vascular, circulatory or permeability deficiencies. The fat emulsion may coarsen and the larger particles may be deposited between the lens fibers, interfering further with the diffusion of fluid through the lens interfibrillar system and into the lens fibers.

INCIDENCE AND TYPE OF OPACITY OF THE CRYSTALLINE LENS IN EIGHTY-EIGHT DIABETIC PATIENTS

A consecutive, unselected series of diabetic patients under treatment was carefully examined to correlate the ocular findings with the general condition. The age of these patients varied from 30 to 77 years, with eight (9 per cent) in the fourth decade of life, thirty (34 per cent) in the fifth, twenty-three (26 per cent) in the sixth, twenty-three (26 per cent) in the seventh and five (5 per cent) in the eighth. Twenty-five per cent of the patients were male and 75 per cent female. The known duration of the disease varied from one to sixteen years. The diabetic condition was classified as very mild in 15 per cent, mild in 24 per cent,

moderate in 35 per cent, moderately severe in 17 per cent and severe in 9 per cent. The blood sugar during fasting varied from 82 to 289 mg. per hundred cubic centimeters of venous blood.

Examination of the Eyes of Diabetic Patients (Under Treatment).—The pupils were dilated with homatropine hydrobromide 1 per cent. After the examination, a drop of 0.5 per cent physostigmine sulphate was instilled. The cornea was examined with oblique illumination; the media, with transmitted light. Particular observation was made of the lens of the eye with the plane mirror at a distance and also with the + 16.00 or + 20.00 lens. The disk of the optic nerve, the retina and the vessels were then carefully observed. The slit-lamp microscope was not used, as it was desired to bring this phase of the work within the scope of the internist.

The Cornea.—The corneas were clear in 66 per cent of the cases, while there was a slight arcus senilis in 15 per cent, a moderate arcus in 12 per cent and a marked arcus in 7 per cent. The diabetic patient showed no greater tendency to this formation than the average patient of the same age group.

The Crystalline Lens and Cataract.—The crystalline lens was clear and free from any objective involvement in 36 per cent of the patients. The remainder (64 per cent) showed various types of cataracts, greatly in excess of the average. The location of the opacity was in the anterior and posterior subcapsular cortex in 2 per cent, in the senile cortex in 70 per cent, in the posterior cortex in 7 per cent and in the nucleus in 21 per cent. In 47 per cent of the patients with involvement of the senile cortex, vacuoles or globules were found in the cortex; in 14 per cent, punctate opacities; in 23 per cent, early water-splitting of the sutures, and in 5 per cent, separation of the lamellae. In the others, the cataract had progressed to the immature or the mature stage; in 2 per cent there was a mature senile cortical cataract in one eye only, and in 10 per cent there were immature cortical opacities. The important findings are that in the greatest percentage of diabetic patients the cataract assumes the form of a senile cortical cataract, and that true diabetic cataract is rare.

In studying the incidence of involvement of the lens in relation to the degree of severity of the disease, changes were found in 40 per cent of the very mild cases, in 66 per cent of the mild cases, in 66 per cent of the moderate cases, in 50 per cent of the moderately severe cases and in 50 per cent of the severe cases. Opacities of the lens were found in 70 per cent of the diabetic patients who showed in addition a recognized condition of general vascular hypertension.

When studied according to the duration of the diabetic disease, changes in the lens were found in the following percentages of the patients in whom diabetes was known to have existed for the period

given: 22 per cent, one year; 62 per cent, two years; 44 per cent, three years; 63 per cent, four years; 40 per cent, five years, and 70 per cent, more than five years.

The Vitreous.—Examination of the vitreous showed it to be clear in 70 per cent of the diabetic patients; of the remainder, 9 per cent showed a slight haze due to fine vitreous opacities, and 10 per cent a greater haze due to the presence of filmy membranes. In no case was there a hemorrhage into the vitreous. A few patients with fluid vitreous were seen. Opacity of the vitreous was not found to be an important factor in this series of diabetic patients.

The Blood Vessels.—Particular attention was paid to the condition of the retinal blood vessels. In determining the presence of sclerosis the following observations were made: 1. Indentation of the veins by the arteries at the points of crossing was present in slight degree in 12 per cent of the cases, in moderate degree in 56 per cent and in marked degree in 14 per cent, a total of 82 per cent of all the cases. 2. Irregularity of the caliber of the column of blood, indicating irregularity of the lumens of the vessels, was observed in 56 per cent. 3. Constriction or narrowing of the caliber of the arteries was observed in 63 per cent. 4. Tortuosity of the vessel was present in 45 per cent. 5. Increased intensity of the light-reflex from the column of blood in the vessels was seen in 41 per cent of the patients. It was found that the latter phenomenon was not a true guide to the presence of sclerosis or thickening and hardening of the walls of the blood vessels, as the intensity of the reflex was great in certain mild cases of sclerosis, while in certain severe cases the reflex was very dim owing to opacity of the media. The other signs as given in the foregoing order were found to be more constant and reliable in the linking up of the retinal vascular picture with the general vascular condition.

There was evidence of general vascular disease with hypertension in 38 per cent of the patients. In these cases, the following observations were made: 1. Indentation of the veins by the arteries at the points of crossing was present in 90 per cent, to a slight degree in 10 per cent, to a moderate degree in 64 per cent and to a marked degree in 16 per cent. 2. Irregularity of the caliber of the vessels was present in 85 per cent. 3. Constriction or narrowing of the arteries was shown in 73 per cent. 4. Tortuosity of the vessels was present in 51 per cent. 5. An increased light-reflex was evident in 49 per cent. 6. Increased fulness of the veins was present in 19 per cent of the cases.

The importance of the signs of vascular sclerosis followed the same sequence in the cases in which hypertension was a part of the condition of general arteriosclerosis. The signs were present to a more marked degree and in a greater percentage of the patients. This emphasized the fact that arteriosclerosis, both general and local, in the eye is an

important complication or coincidence in diabetes, and that many ocular lesions may be ascribed to the vascular disease rather than to the diabetes.

The Retina.—In the study of all the cases of diabetes, 50 per cent were found without ophthalmoscopic evidence of retinal lesions. Hemorrhages were found in 21 per cent, exudates in 14 per cent and edema in 15 per cent, while changes secondary to previous hemorrhages and exudates were evident in 12 per cent. Healed choroidal lesions were observed in 3 per cent, and atrophy of the optic nerve and detachment of the retina, in 1 per cent. The fundus was not visible because of opacities of the lens in 1 per cent. The severity of the disease did not seem to influence the incidence of retinal lesions, as they were present in 40 per cent of the very mild cases of diabetes, 45 per cent of the mild cases, 46 per cent of the moderately severe cases and 38 per cent of the severe cases in which treatment was given. Retinal lesions were present in 55 per cent of the cases complicated by vascular hypertension. Hemorrhages were found in 21 per cent, exudates in 19 per cent and edema in 12 per cent, while changes secondary to previous hemorrhages and exudates were seen in 24 per cent. It was in the last respect that the cases complicated by vascular hypertension differed from the general case of diabetes in this series; that is, the retinal lesions secondary to previous lesions were more frequent.

Other Complicating or Incidental Diseases.—Focal infection was noted in 9 per cent of the diabetic patients, while arthritis, rheumatism or neuritis was present in 19 per cent, myocarditis in 14 per cent, syphilis in 8 per cent and tuberculosis in 1 per cent.

Coincidence of Changes in the Lens (Incipient Cataract) and Retinal Lesions in Diabetes.—Retinal lesions in the form of hemorrhages, exudates or edema or changes secondary to these were found in 40 per cent of the diabetic patients whose lenses were clear, while such lesions were found in 53 per cent of those with incipient cataract. A greater proportion of the diabetic patients with incipient cataract than of those with clear lenses have retinal lesions, although such lesions do not necessarily accompany the formation of cataract. However, a great number of diabetic patients with advanced cataract have probably suffered from retinal lesions.

Uhthoff and Arruga were impressed with the occurrence of other ocular complications of the diabetic condition. They reported a fair percentage of cases marked by the presence of retrobulbar neuritis, paralysis of the ocular muscles, hemorrhagic glaucoma and iritis. I have seen such complications in diabetic patients, but not in this series. The patients in my series were all under active treatment, and this may account for the lesser number of complications. Lipemia retinalis has

also been seen. In only one case have I seen the condition described as rubeosis iridis, with vascularization and thickening of the anterior portion of the pupillary area of the iris. The hypotonia seen uniformly in diabetic coma was not observed in the ambulant series.

CATARACT IN AN ADOLESCENT DIABETIC PATIENT

R. S., a boy, aged 16, with diabetes, was under treatment for eighteen months. On admission to the hospital, the blood sugar during fasting was 279 mg. per hundred cubic centimeters of blood. The condition was apparently well controlled, and the patient was under treatment with insulin when the posterior subcapsular opacity of the lens began to develop. This reached the immature stage within four weeks, reducing vision below the useful range. The features in the lens as observed with the aid of the slit-lamp microscope were subcapsular vacuoles or globules, water-split sutures and cloudy swelling of the cortex. The cataract in one eye was removed by discission followed by linear extraction in one week, with good results. Vision with a correcting lens was 20/20. No evidence of retinitis was found.

PREPARATION OF DIABETIC PATIENTS FOR OPERATION ON THE EYE

The optimum condition for the diabetic patient prior to an operation is that in which healing is promoted, infection resisted and any tendency to acidosis removed. The degree of well-being in the patient will be enhanced if the blood sugar can be reduced to approximately normal levels either by restriction of the diet, if this is accomplished easily, or by the addition of insulin. It has been our experience that the patients are in better condition if the intake of carbohydrate is sufficiently high, insulin is used and the consumption of fat is low than if the carbohydrate is unduly restricted to keep down the blood sugar without the use of insulin, while the fat is increased to meet the caloric requirements. Cooperation with an internist is necessary. The fundi, if visible, should be carefully studied, and the intra-ocular tension should be taken. There are three points worthy of note in the reaction of the eye of a diabetic patient to an operative procedure: (1) the tendency to hemorrhage because of the fragility of the sclerosed vessels, (2) the difficulty of properly dilating the pupil and (3) the exfoliation of the pigment granules due to rupture of the edematous vesicles of the pigmented epithelial layer of the iris and ciliary body.

SUMMARY AND CONCLUSIONS

My experience is in accord with that of Duke-Elder in that there are two forms of cataract which occur in diabetes: (1) the form of senile cataract appearing frequently and at a relatively early age and (2) a rapidly developing form peculiar to the disease occurring more rarely.

The changes in the focus of the eye are most probably due to alterations in the osmotic pressure of the ocular fluids affecting the crystalline

lens. This is also true in part for the production of opacities of the lens. There may be other factors little understood at present which are responsible.

Opacities of the lens were found in 64 per cent of a group of diabetic patients of all ages under treatment examined consecutively. Of these, 70 per cent had the opacities in the senile cortex. The severity of the diabetes did not influence the incidence of the opacities. The duration of the disease had a bearing, as the greatest percentage of cases in which opacities were present was found in the patients in whom the disease had existed for more than five years. Opacities of the lens were found in 70 per cent of the diabetic patients who showed in addition a recognized condition of general vascular hypertension. It was found that arteriosclerosis, both general and local, in the eye is an important complication or coincidence in diabetes, and many of the ocular lesions could be ascribed to the vascular disease rather than to the diabetes.

Retinal lesions in the form of hemorrhages, exudates or edema or changes secondary to these were found in 40 per cent of the diabetic patients under treatment whose lenses were clear, while such lesions were found in 53 per cent of those with incipient cataract. A greater proportion of diabetic patients with incipient cataract than of those with clear lenses have retinal lesions, although such lesions do not necessarily accompany the formation of cataract. However, the greater number of diabetic patients with advanced cataract have suffered from retinal lesions.

Clinical Notes

AN IMPROVED ILLUMINATOR AND PUPILLOMETER

J. B. Feldman, M.D., Philadelphia

In February, 1932, I [1] reported an improved campimeter, in which the illumination could be controlled at 7 foot-candles or less when necessary. The use of the control instrument for checking up on the illumination proved to be the most difficult feature in the operation of the instrument. This was accomplished by comparing both the central and the movable stimuli of the campimeter with a standard of illumination, consisting of an incandescent lamp and filter, a source of current (dry cells), a rheostat for controlling the current and a milliameter for indicating the intensity of the current. This control instrument was originally calibrated against a Macbeth illuminometer. Two factors which proved troublesome in the check of the illumination of the campimeter were: 1. The cells of the control unit were usually dead just when the device was needed. 2. The test at best was subjective and therefore not very reliable.

To obviate these uncertainties an illuminometer was devised, utilizing the Weston photronic cell. This cell was used for two reasons: 1. It requires no source of electromotive force for its operation. 2. Its response to light resembles very closely that of the human eye.

The commercial form of illuminometer, utilizing the photronic cell manufactured by the Weston Company, could not be used because the aperture for exposure was limited to 6 mm. by the requirements of the campimeter.

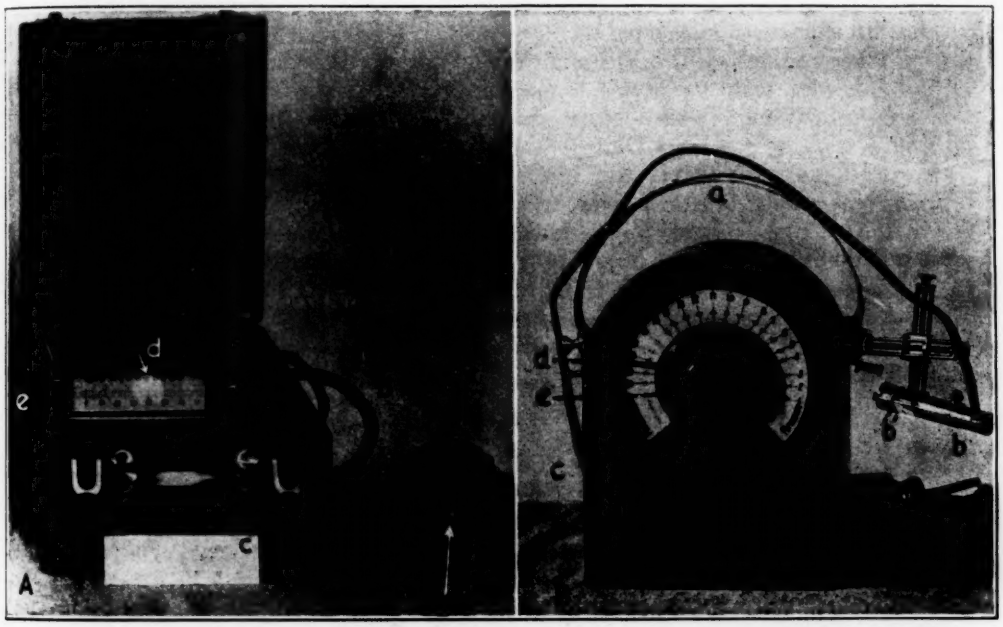
The apparatus was finally developed as pictured in figure 1 *A*. In use the aperture (*b*) is exposed to the light under investigation and the reading is indicated quickly and accurately on the graduated opal glass *c* or *c'*. Readings up to 20 foot-candles are obtained with the instrument as shown and with scale *c* in position. Readings up to 40 foot-candles and to 110 foot-candles are obtained by the insertion of one of two required shunts across the galvanometer and the use of scale *c* or *c'*. The process is simple and objective, and no special skill is required. When necessary, as in my perfection of the pupillometer, the photronic cell is substituted for the human eye to record the quantitative stimuli of the light emitted by the Sander Pupilloscope.

In 1916, Hess [2] described a pupilloscope the purpose of which was to indicate qualitatively the direct, consensual reaction as well as the threshold value of the light reflex, both motor and visual, known respec-

Read before the Section on Ophthalmology, College of Physicians and Surgeons, Philadelphia, Jan. 19, 1933.

1. Feldman, J. B.: M. J. & Rec. **135**:197 (Feb. 17) 1932.
2. Hess: Arch. f. Augenh. **80**:213, 1916.

tively as motor and visual discriminative acuity, a useful agent in the early diagnosis of the Argyll Robertson pupil. His work was augmented by Groethuysen [3] and popularized in America by Barkan,[4] Adler [5] and Gifford.[6] Sander devised a modification of this instrument which was cheaper and could be used with the slit-lamp. This instrument has, however, several objectionable features; i. e., the results are also qualitative; like the Hess apparatus, the stand must either be a slit-lamp stand or some similar device; the shutter movement produces a click disturbing to the patient, and furthermore it is difficult to obtain single notch movements of the "Goldberg wedge" (single graduations of light).



A, illuminometer: *a*, Weston Photronic Cell, encased in a supporting holder; *b*, 6 mm. aperture in the mask of the photronic cell; *b'*, leads connected to a sensitive galvanometer; *c* and *c'*, special engraved scales calibrated in foot-candles from data obtained from a test of the illuminometer by the Electric Testing Laboratories; *d*, spot of light indicating quantity of illumination; *e*, sensitive galvanometer. *B*, pupillometer: *a*, headband; *b*, holder; *b'*, gage; *c*, transformer; *d*, dial; *e*, push-button.

In order to translate the arbitrary readings of the Sander pupilloscope (21 to 1) into quantitative values of illumination, the photronic cell was fixed rigidly in the head rest of the slit-lamp, with the aperture

3. Groethuysen: Arch. f. Augenh. **87**:1521, 1921.
4. Barkan: Arch. Ophth. **51**:29, 1922.
5. Adler: Arch. Ophth. **55**:262, 1926.
6. Gifford, S. R., and Mayer, L. L.: The Clinical Use of the Sander Pupilloscope, Arch. Ophth. **6**:63 (July) 1931.

(*b*) of figure 1 *A* occupying the same position as the pupil of the patient to be examined.

Each individual reading from 21 to 1 on the Sander instrument was translated into foot-candles by the readings on the illuminometer (fig. 1 *A*). This was rechecked by several observers.

With these values determined, a simple, portable, inexpensive pupilloscope, better called a pupillometer or pupilloscope-meter (fig. 1 *B*), was devised. The headband (fig. 1 *B*, *a*) for the patient contains a holder (*b*) which carries a source of illumination set at a fixed distance from the patient's eye by gage *b'*. The holder *b* can be moved in all directions and can be quickly removed from the headband (*a*) in an emergency and used by itself. The lamp in *b* is controlled by a rheostat and a transformer in box *c*, which, in turn, is connected to the alternating house current.

The reading of the illumination is indicated by a moving spot of light on the scale of box *c*. The outer rim of the scale is graduated into the arbitrary pupilloscope readings (1 to 21) and the inner rim of the scale is graduated into corresponding foot-candles (fig. 1 *B*).

The dial *d* controls the intensity of illumination and through its center is a push-button *e*, which, when pressed down, produces in every case, regardless of dial *d*, the lowest possible illumination of the instrument, equivalent to the upper portion of the Goldberg wedge. Release of the push-button *e* returns light to the quantity indicated on the dial The change is silent and instantaneous.

The Wellsworth Beebe Loupe is used by the observer. This entire instrument has been found both cheap to construct and very reliable.

The Weston Electrical Instrument Company permitted the use of their photronic cell for these and other experiments to be undertaken.

A NEW TECHNIC FOR THE PREPARATION OF PATHOLOGIC SPECIMENS FOR MICROSCOPIC STUDY

PERCE DE LONG, M.D., PHILADELPHIA

Numerous methods have been described for the preparation of pathologic material for microscopic study, but all are not efficacious for ocular material because of the difference in density of the ocular tissues. Frequent experimentation has revealed varied results, with the usual return to the common method of embedding in celloidin.

I have tried every process suggested to me, including the dry method with the block prepared in cedar oil, the slow method which requires from four to six weeks for the infiltration of the celloidin, the hot celloidin method and many others, all of which present disadvantages.

The most satisfactory method that I have employed is the one suggested by Prof. George de Renyi of the University of Pennsylvania.

Read before the Section on Ophthalmology, College of Physicians of Philadelphia, Nov. 17, 1932.

With modifications, I have found it the most practical because of the saving in time, the thinness of the sections and the manner of handling the block, which does not require constant preservation in alcoholic solution.

Of the various fixatives, Müller's fluid is best, as it causes less shrinking of tissue and therefore fewer artefacts, but it requires two weeks for proper fixation, which is a disadvantage.

Orth's solution, Zenker's solution, a 10 per cent solution of formaldehyde and Bouin's solution are excellent for fixation, but none has proved as satisfactory as Susa's fluid, which is composed of mercuric chloride 45 Gm., sodium chloride 5 Gm., and distilled water 700 Gm. These ingredients are boiled for five minutes and cooled, and the following are added: trichloracetic acid (20 per cent), 100 cc.; formaldehyde, 200 cc., and acetic acid, 40 cc. The time required for fixation is from six to fifteen hours, depending on the size of the specimen. A globe requires about twelve hours.

Before the specimen is placed in the solution, a small niche should be made into the anterior and vitreous chambers so that the solution may have access to them. The specimen when fixed is removed at once to 95 per cent alcohol. It is not washed, because trichloracetic acid has been used and water would therefore have a deleterious effect on the connective tissue.

After the specimen has been placed in 95 per cent alcohol, 2 drops of iodine are added. Soon it is noted that the iodine tint has disappeared, the alcohol solution having returned to its normal color. Two drops of iodine are again added, and this procedure is repeated until the iodine color remains in the alcohol.

The eye is washed in 95 per cent alcohol to remove the "back wash" of the iodine. It is then placed in 95 per cent alcohol for twenty-four hours, in absolute alcohol for twenty-four hours, and finally in equal parts of absolute alcohol and ether for twenty-four hours.

It is worthy of note that here there is a saving in time of dehydration of three days, as the twenty-four hours of washing in 70 per cent, 80 per cent and 90 per cent alcohol has been eliminated.

The specimen is now ready for embedding. It is placed in 2 per cent celloidin for forty-eight hours, in 4 per cent celloidin for forty-eight hours and finally in 6 per cent celloidin (no higher concentration) for forty-eight hours. It is then placed in a small receptable coated with paraffin and is hardened by exposure to chloroform vapor until the celloidin has the consistency of hard rubber. The absence of air bubbles in the celloidin is a decided advantage.

The excess celloidin is pared away, and the desired block is transferred to a solution of benzene for twenty-four hours, to a second solution for twenty-four hours, and, to a third for twenty-four hours. The specimen is then free from cloudiness. The block has the appearance of crystallized candy encasing the specimen. It is now ready for the double embedding in paraffin.

The block is placed in paraffin in an oven heated to 56 C. (never higher) for twenty-four hours, in a second preparation of paraffin for twenty-four hours, and in a third for twenty-four hours. It is now ready to block and can be left indefinitely for cutting. It is not necessary to keep the block in alcohol or cedar oil.

The knife is cleaned with alcohol and dried, and egg albumin is applied to its surface. It is then floated with 30 per cent alcohol (never stronger), and the specimen is cut in the usual manner, with a slanting knife. I have found the Reichert microtome the most satisfactory.

Depending on the keenness of the knife edge, one will be surprised to find that sections can be cut as a routine at 7 millimicrons, and at as low as 4 millimicrons, according to the density of the tissue. Liver has been cut as low as 2 millimicrons. If this thickness is compared with the older ones of 13 millimicrons, formerly considered satisfactory, one can at once see the advantage of this method of preparation.

After the sections are cut they are placed in distilled water and put in an incubator at 38 C. for twelve hours. This is necessary in order to remove the wrinkles, which are difficult to iron out, and probably constitute the only unfavorable feature of this method. The sections are now ready for staining and mounting.

SIMPLE AND INEXPENSIVE METHOD FOR MOUNTING EYES

B. F. PAYNE, M.D., HOUSTON, TEXAS

A simple process for mounting eyes is as follows: After enucleation the globe is transilluminated and placed in a diluted solution of formaldehyde (5 per cent). At the end of four or five weeks, it is removed, washed under the tap and divided longitudinally to show the pathologic changes to the best advantage. The eye is fixed between the fingers on a small wooden block and severed with a (Durham-Duplex) razor blade held by a small hemostat. Sufficient care is taken to preserve as much of the globe as possible for sections, should they be desired at a later date. Occasionally, the vitreous has formed a jell from the action of formaldehyde.

The globe is fixed at the center of an ordinary microscopic slide by means of a white silken suture. The slide is selected rather than the opaque supports generally used, in order that the specimen may be seen from all angles. The specimen and slide are inserted into an ordinary round bottle, the outside measurements of which are 4 cm. in diameter and 8.5 cm. in length; this is easily obtained from any pharmacist. A thin cork is notched to support the upper end of the glass slide in the position desired. The bottle is filled with a diluted solution of formaldehyde (5 per cent), the cork inserted and the bottle sealed by dipping the top into a crucible of melted sealing wax. The rough edges are smoothed with an alcohol flame and heated spatula.

The specimens are numbered, and a drawing corresponding to the eye, as well as a brief history with the "findings" is placed in a special book.

The advantages of such a procedure are:

1. It provides complete visibility of the specimen from practically all angles.

2. It furnishes a simple and inexpensive method for preserving pathologic specimens.
3. Special skill is not required.
4. More globes will be sectioned for microscopic study owing to questions evolved from gross examination.
5. An orderly and complete record is provided for sections to be made later.

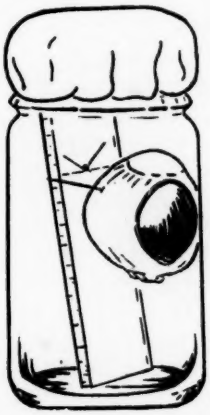


Fig. 1.—Drawing illustrating the completed mount as described in the article.

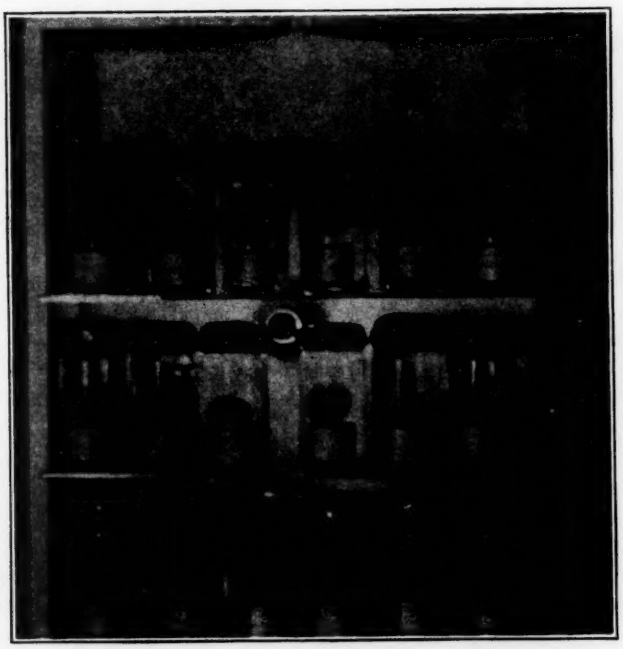


Fig. 2.—Photograph showing specimens arranged on the glass shelves of a cabinet with central illumination (glass door open).

FRACTURE OF THE MAXILLA THROUGH THE LEFT OPTIC FORAMEN

Reduction with a Kingsley Splint, with Restoration of Vision

H. MAXWELL LANGDON, M.D., PHILADELPHIA

Miss H. M., aged 23, was admitted to the Presbyterian Hospital to the service of Dr. John Speese on May 31, 1932, immediately following an accident to the motor car which she was driving.

When admitted she was semiconscious, with a badly injured head and face, and was bleeding from the nose and mouth. She was given emergency treatment in the receiving ward. Her head was examined roentgenologically and she was then admitted to a private room. She soon recovered consciousness, but was in a state of moderate shock, the temperature being 95 F. and the blood pressure 100 systolic and 70 diastolic. There was no bleeding from the ears. Some lacerations were seen under the left eye, near the right angle of the mouth. The entire face was badly contused and swollen. The eyelids, being ecchymotic and swollen, could not be opened naturally. The conjunctivae were swollen; there was a deep indentation over the bridge of the nose, and the nose itself was considerably flattened, blood coming from both nostrils. The lips were swollen and the lower jaw seemed to be displaced. Blood was slowly coming from the mouth, which could be opened only a slight distance with difficulty; there was a definite crepitus of the right side of the mandible. A contusion of the left shoulder and some cardiac arrhythmia were present.

She was treated for the shock and was seen by Dr. Cariss and Dr. Aiguier. The following day I was asked to make an examination of her ocular condition. The swelling of the lids had somewhat subsided, and the right eye seemed normal in all particulars. The left eye showed a sluggish and slight reaction of the pupil to light, and vision was diminished to perception of light, with, however, no changes in the fundus. A diagnosis of fracture through the orbit, involving the optic foramen, or a probable hemorrhage into the sheath of the nerve was made and a poor prognosis for vision of the left eye was given.

Dr. Cariss found that there was a displaced fracture of the nasal bone, almost completely blocking the nostrils, with a rupture of the mucous membrane of the septum and the left ethmoid region, from which the hemorrhage was coming. This was controlled by packing. Three weeks later the nasal bones were straightened under local anesthesia, making it possible for the patient to breathe comfortably through the nose after removal of the packing. The roentgenogram showed multiple lines of fracture crossing the left parietal and temporal bones. There was also a line of fracture through the ethmoid sinus across the bridge of the nose and through the right mandible.

Dr. Aiguier's examination showed a fracture of the maxilla running through the orbit and sloping down on either side in the molar region, with the maxilla movable in any direction. There was also a fracture in the right third molar region and a fracture of the left condyle just above the notch. On June 6, another ocular examination showed that the conditions were as on the first examination, with the exception of beginning pallor of the left disk.

Dr. Aiguier took impressions of the teeth and made an upper cast, Kingsley splint and a lower splint that could be fixed in proper occlusion by lugs with plaster of paris, with attachments for fixation. There was a slight elevation of

temperature most of the time for the first three weeks that the patient was in the hospital. During the first week it reached 100.5 F. on two occasions. After this it remained between 99 and 100 F. until about a week before the patient's discharge, when it returned to normal.

When the splint was put in place, pressure was applied gradually, as it was feared there might be greater damage to the nerve as the parts adjusted themselves. There seemed to be slight improvement in vision almost at once, and this continued gradually until, within two weeks after the splint was first adjusted, the vision had improved to about 5/45. This was tested roughly in the room. The improvement continued, and the first time that the patient was taken to the eye dispensary for an accurate test, vision was found to be 6/20, with a paracentral scotoma to the lower temporal side of fixation. This scotoma was partially relative and partially absolute, the absolute portion becoming smaller as time went on. The central vision gradually improved until it is now 5/12. The disk is still pale and the scotoma still persists. The patient has made an excellent recovery in all respects. A slight deformity of the nose is to be corrected later by operation.

Fracture of the anterior portion of the skull, involving the orbit and the optic foramen, usually results in the blindness of the eye through hemorrhage into the sheath of the optic nerve or atrophy of the nerve from pressure, and a poor prognosis is almost invariably given. The experience with this case shows that when there is a fracture in the lower portion of the orbit involving the maxilla, mechanical intervention may relieve the pressure and bring about a restoration of function; should the roof of the orbit be involved, however, it seems much less likely that intervention will be successful in the restoration of vision, as replacement of the fractured fragments in this part of the orbital wall is obviously most difficult of accomplishment. It would seem that the proper proceeding would be to make a careful roentgen examination. with accurate determination of the line of fractures before too unfavorable a prognosis is justified. In the case here reported the disk is very pale from the atrophic process, but useful visual function has been preserved.

Ophthalmologic Review

PRESENT STATUS OF SURGICAL TREATMENT OF RETINAL DETACHMENTS

MARK J. SCHOENBERG, M.D.
NEW YORK

Professor Gonin reported a series of 300 cases of retinal detachments in which the patients were operated on by his method. His conclusions, being based on a large number of cases observed over a period of more than ten years, are naturally quite acceptable. With this series he reaffirmed his original conception that a tear or hole in the retina is the cause of the detachment, and that in recent cases the occlusion of this opening is sufficient to bring about a complete cure. A cure is obtained in 55 per cent of cases if treatment is applied within three weeks of the onset of the illness. In 15 per cent of Gonin's cases it was necessary to repeat the procedure, and the results were satisfactory. Of all the cases with a detachment of less than one year's duration, cures were obtained in 40 per cent. A recurrence of the detachment occurs if the occlusion of the tear is insufficient or if a new tear appears. Recurrences are curable by the same treatment as the original detachment. Insufficient attention to the localization of the tears is a frequent cause of failure.

THE SOURDILLE METHOD

Parallel with Gonin's work, Sourdille pursued the problem of surgical treatment of retinal detachments along principles somewhat different from those of Gonin. Sourdille's idea is that the main pathologic process takes place in the vascular system of the choroid, principally in the choriocapillaris. This disturbance causes a separation between the retina and its pigment epithelium layer and an accumulation of fluid in the space thus created. The object, then, of the treatment is to evacuate the fluid as completely as possible and to cause a reaction in the choroid which should result in an adhesion between it and the retina. To obtain this effect Sourdille uses a cautery in some cases and punctures with the Graefe knife in most of the cases; in a few others he uses both procedures. He advises the use of punctures with the knife in detachments of high elevation (blebs, balloons and the like), while he limits the indication for the use of ignipuncture only to those

cases in which there are flat detachments. He does not believe that it is essential to cauterize the margins of the retinal tear.

The details of his operation are as follows: 1. A few days before the operation, absolute rest in bed is necessary, during which time calcium chloride is given to prevent choroidal hemorrhage. 2. Sclerochorioretinal punctures are performed like any ordinary posterior sclerotomy. The number of punctures depends on the extent of the detachment. In total detachments Sourdille makes four punctures, placed midway between the four recti muscles from 15 to 18 mm. behind the limbus corneae. 3. After the termination of the operation he injects a few minims of 1:1,000 solution of mercury oxycyanide under the conjunctiva of the punctured areas. 4. An atropine morphine ointment is used in the conjunctival sac, and a binocular bandage is applied. 5. Absolute rest for from fifteen to twenty-one days is necessary.

It is often necessary to repeat the treatment several times. Of 170 patients operated on, 79 had a good result. Some of the patients remained under observation for from eight to ten years. Sourdille enumerates the advantages of his method as follows: It is applicable to patients without a tear, and it does not cause an extensive injury to the retina or choroid. He insists on the general treatment of the patient, with the object of improving the general circulation and renal elimination.

The conception underlying the Gonin method has been adopted by a large number of ophthalmologists from all over the world, and the literature of the past five years contains numerous reports of successes and failures. Many of the failures have been due to insufficient knowledge of and attention to the operative indications and technic. None of the eye surgeons have been able to duplicate the good showing of Professor Gonin's statistics. Even his own assistant, Dr. Amsler, could not obtain more than 50 per cent of cures in 40 patients operated on, although he was cautious in selecting them. Of 115 patients, Amsler operated on only 40.

In the introduction to his paper, Gonin enumerates the types of patients who are not suitable for his method as follows: (1) those with opaque or semiopaque media; (2) those with tears that are too large or are situated too far posteriorly; (3) those with too many tears, and (4) patients who are unable to keep quiet after the operation so as to insure an adhesion between the choroid and the retina. He does not mention those with a detachment without a tear. The numerous failures of ophthalmologists who have tried to master the technic of Gonin's method and the large number of patients they had to reject as not suitable for this operation have stimulated them to search, first,

for definite operative indications and, second, for a modification of the method which would make it possible to obtain a larger number of successful results.

Blatt divided detachments of the retina into two classes: (1) the exudative type due to nephritis, syphilis, tuberculosis, diseases of the orbit and nasal sinuses, pregnancy, eclampsia and disturbances of the circulation, which should be treated along medical lines, and (2) the idiopathic type found with myopia and senility and also traumatic cases. These conditions do not heal spontaneously, nor do they respond to conservative treatment. For these cases Blatt advised the Gonin operation.

Lindner went one step further, and tried to determine, from a study of the condition of the vitreous, the operative indications and the proper time for surgical intervention. He said that when the retinal bleblike detachment presents fine ripples on its surface and does not change its shape and position during sudden movements of the eye, or while the patient is placed in a recumbent position, the vitreous adjoining the retina is of a jelly-like consistency and is adherent to the retina. On the other hand, when the detached retina has a smooth surface, floats with ease, flattens out considerably when the patient lies quietly on his back or changes its position when the patient lies on either side, the vitreous is fluid and contains no strands which may pull the retina away from the choroid. Lindner's conclusions are that in many cases it is preferable to wait until the retinal bleb is free from adhesions to the vitreous, and this takes place after its fluidification. Lindner's views have not been verified entirely by others. These attempts of establishing operative guiding posts, though by far incomplete, should be of some help to those who intend to familiarize themselves with the complex problem of surgical treatment of retinal detachments. Lindner found a tear in 84 out of 118 cases, and, like Gonin, he believes that there is a causal relationship between the tear and the detachment of the retina. He thinks that the disappearance of the detachment after the occlusion of the tear is the strongest evidence in favor of this relationship. During the preiod from 1928 to 1930, he operated, according to the Gonin method, on 61 patients with retinal tears, and was successful in 23; 5 of 21 patients with detachments without discoverable tears were cured. Some of the more serious disadvantages of the Gonin method are: (1) necrosis of the retina adjoining the ignipuncture; (2) injury to the vitreous and formation of traction strands in the vitreous; (3) formation of scars in the retina, which tend to displace it and cause new tears and detachment; (4) hemorrhages into the vitreous. In 3 of Lindner's cases the eyeball became atrophic, and in 1 the eye was lost because of panophthalmitis.

THE GUIST METHOD

The complications, the relatively small percentage of cures, the difficulty of localizing the tear and at times the impossibility of finding a tear, as well as the hopelessness of detachments without a tear, led Guist and Lindner to employ another method. In 1930 they began to use a method devised by Guist and reported in that year. The underlying principles and purpose of this method are as follows: (1) to produce an inflammatory reaction in the choroid and the detached portion of the retina and thereby to obtain an occlusion of the tear; (2) to isolate by a line of (inflammatory) demarcation the entire area of retina which is detached from that which is intact.

The Guist method, as described by Lindner, consists of the following steps:

1. A retrobulbar injection of 1 or 2 cc. of 2 per cent solution of procaine hydrochloride, to which a few drops of a 1:1,000 solution of epinephrine has been added, is made.

2. The conjunctiva is incised parallel with the limbus corneae, near the fornix, and the sclera is exposed over the entire area corresponding to the detached retina, if it does not exceed one half of the fundus. The operative field is made accessible by retractors, sutures through the tenon capsule and tenotomy of one or even two recti muscles.

3. The exact localization of the tear or tears of the retina, as well as of the borderline up to which the retina is detached, is marked on the sclera with the aid of the electrocautery or blue pencil.

4. A number of trephinings, 1.7 mm. in diameter, are performed, first along a line circumscribing the tear area, and, second, if possible, along the line of demarcation between the detached and the nondetached retina. If one is dealing with a dialysis at the ora serrata, the trephinings are applied along a concave line, parallel and just outside the arc of the dialysis. The trephine openings should not go right through the last thin layer of sclera, for fear of perforating the choroid, which might be atrophic in some places. This thin layer of sclera is cautiously removed with the point of a small, sharp keratome. The trephine openings are placed at a distance of not more than from 1.25 to 1.5 mm. apart, since the effect of the chemical cauterization of the choroid does not spread over the surrounding area by more than 0.5 mm. If the retina is very much elevated and the detachment is extensive, one should apply a number of trephinings over the rest of the exposed sclera as well.

5. After the choroid has been exposed, it is cauterized with the tip of a potassium hydrate pencil for not more than one second. This area is neutralized immediately by a 0.5 per cent solution of acetic acid

followed by a gentle irrigation with salt solution. The cauterization, neutralization and irrigation can be applied to three or four choroidal areas at one time.

6. When the cauterization has been completed, the choroid is perforated in a number of places so as to give escape to the subretinal fluid and to allow the retina to come in contact with the choroid. If the number of trephinings is small, one should perforate each trephine opening; if the number is large (between 15 and 30), the perforations should be limited to about one-third. One should avoid placing a trephine opening near a vena vorticosa, and one should not perforate the choroid where a large vessel is in evidence.

The tenotomized muscles are reattached, and the conjunctiva is sutured only after the hemorrhage has been entirely arrested.

The after-treatment is somewhat similar to that following a Gonin operation: absolute rest in bed for three days, with the head in the "Gonin position" (the vitreous should rest on the portion of the retina with the tear, if possible). The binocular bandage is removed on the eighth day, and frames with stenopeic diaphragms are worn for the following six or seven weeks. The patient is allowed to sit up quietly from one to two weeks after the operation.

Lindner reported in his paper the details of each case and the results. Of 19 patients, 13 were cured. It takes about three or four months for the reaction of the retina and choroid to reach its end. There is no formation of traction scars or necrosis of the retina after this operation. Hemorrhage can be prevented by avoiding the large vessels, especially the venae vorticosae. The procedure is lengthy (from one to two and one-half hours), and at times the trephine perforates the choroid at the beginning of the operation; collapse of the eyeball due to the escape of vitreous makes continuation of the operation impossible.

According to Guist and Lindner, the percentage of cures is much higher than that obtained by the Gonin method, and, in addition, even some patients unsuitable for the ignipuncture method stand a chance of being cured by the Guist procedure.

At the 1932 meeting of the German Ophthalmological Society, Guist reported his own series of cases. Of 61 patients with noncomplicated cases, 47 were cured (77 per cent). In 11 complicated cases (iridocyclitis, retinitis proliferans and vitreous hemorrhages) he obtained 2 good results; of 7 patients with cases of four months' duration, who had been treated unsuccessfully with the Gonin method, 4 were cured, and of 17 patients with cases of eight months' duration who had previously been treated by ignipuncture, 2 were cured by the Guist method. In 13 aphacic eyes with detachment, he obtained 5 cures. The average of cures amounted to 56 per cent.

Guist stressed the point that it is essential to occlude the hole in the retina, and that it is of equal importance to establish an adhesion along the borderline of the periphery of the detached retina. He advised that the treatment should be applied over the entire area, at least from eight to 17 mm. behind the ora serrata. Lindner reported the following visual results:

8 had a vision of	6/6
5 had a vision of	6/9
3 had a vision of	6/12
9 had a vision of	6/18
1 had a vision of	6/24
1 had a vision of	6/60

Other patients: from 6/18 to counting fingers at 30 cm.

Guist's views regarding the tears and the condition of the vitreous are in agreement with those of Gonin. He saw the tears developing after the appearance of the retinal detachment in areas with cystic degeneration of the retina. In U-shaped tears with tonguelike flaps of the retina, the tear is due either to a bulging sclera or to traction from within by vitreous adhesions and strands. Multiple small holes are due to cystic degeneration of the retina, and the occlusion of the retinal tear and the escape of the subretinal fluid are essential for the reattachment of the retina. The exact localization of the tear is not important, since Guist places the trephine openings over the area corresponding to the tear as well as over the rest of the surface covering the detached retina.

So far there are few reports in the literature on cases in which the patients have been treated by the Guist method. In the United States, Dr. Hugh S. McKeown reported on 51 cases before the American Academy of Ophthalmology and Otolaryngology. He considers as cured patients with the retina attached and vision of 20/100 or better, and as improved those with the retina replaced but without improvement of vision. His results were: 20 cures (39 per cent) and 7 improvements. He encountered the following serious complications: intra-ocular hemorrhages in 5 cases, endophthalmitis in 1 case and hypotomnia in 3 cases.

Lindner summarized the advantages of the Guist method as follows: (1) It does not shorten and scar the retina; (2) it succeeds sometimes even in old cases; (3) the complications are less frequent and less severe than after the Gonin operation, and (4) it does away with the difficulty of exact localization of the tear. The main disadvantages of the Guist method are: (1) the tediousness of the procedure (it takes between one and three hours to complete such an operation); (2) the possibility of perforating the choroid and of losing subretinal fluid at the beginning

of the operation; when this occurs, the softening of the eyeball prevents the application of more trephinings to the sclera, and the operation has to be postponed; (3) the difficulty of avoiding large blood vessels, and, consequently, the relatively frequent occurrence of intra-ocular hemorrhages, and (4) the occasional loss of an eye from infection.

THE WEVE-LARSSON-SAFAR METHOD

The third stage in the evolution of the surgical management of retinal detachments was initiated by Larsson (1930) and Weve, and was modified by Safar.

They conceived the idea of closing the retinal hole in a detached retina by a simpler method, which caused the least damage to the retina and vitreous. For this purpose they employed diathermy. When the inactive electrode of a diathermy apparatus is connected with a plate kept in contact with a large surface of the body and a small active electrode is applied on the surface of the sclera, a weak current of less than 150 ma. is sufficient to produce a coagulation of the underlying tissues, sclera, choroid and retina. The reaction following this treatment is sufficient to produce an adhesive process, which reattaches the retina to the underlying choroid.

Weve, and lately also Larsson, expressed the belief that obstruction of the retinal hole is essential for the success of the operation, but they feel that their method is efficient even in cases without a tear. Disagreeing with Gonin, they insist that the entire area corresponding to the detachment must be treated. Larsson described his method in detail in 1932. The procedure has not been in use a sufficient length of time to allow one to know enough about the final results. So far, Larsson has reported 40 cases in which operations were performed. In 20 (50 per cent) he obtained complete reattachment of the retina to the choroid. He believes that the technic could be improved, that the ampere reading is not a reliable guide of the amount of heat developed within the tissues. The latter depends rather on other factors, such as tissue resistance, moisture and contact. Reattachment of the retina is brought about by the tissue reaction. The resulting atrophy of the choroid may act in a beneficial manner by reason of the fact that the conditions for the formation of fluid from the choroid no longer exist. Larsson thinks that the diathermy method, like the Guist operation, prevents recurrences of the detachment by the mere fact of the adhesions over a large area affected by this method. Hemorrhages into the vitreous are rare owing to the coagulation of the blood vessels; no injury is caused to the vitreous, and no traction strands form. There is no necessity for the exact localization of the retinal hole. Professor Weve of Utrecht has used a method of diathermy which is somewhat

different from that of Larsson. His procedure consists of two steps: (*a*) surface diathermy and (*b*) micropunctures. He employs either procedure, or, at times, he combines both in the same case. One should not overlook his insistence principally on the importance of localization of the tear and on the application of diathermy especially over the area corresponding to the tear. (He still recommends the Gonin method in limited detachments with small tears which are easily accessible.) Weve, with the aid of surface diathermy, obtained good results in retinal detachments with large tears at the ora serrata, but in balloon-like detachments with tears further back, he employs another procedure which he calls micropunctures.

Professor Weve prefers the diathermy apparatus of Monbrun and Casteran (manufactured by Walter of Paris). After the usual preparatory steps are taken—exact localization of the retinal tear, washing the eye, instillations of phenacaine and subconjunctival injection of a 2 per cent solution of procaine hydrochloride to which 1 or 2 drops of epinephrine have been added—a conjunctival flap is prepared in the region corresponding to the area of the detached retina containing the tear. The inactive pole of the apparatus is attached to a cuff (of tin) applied to the arm or to a flat piece of tin about 20 cm. in diameter in direct contact with the back of the patient. The active electrode, previously sterilized, is applied to the sclera. The current is then released through the foot control. The active electrode is a thin metal rod about 1 mm. in thickness with a spherical button at its extremity from 3 to 5 mm. in diameter. The current must not exceed 150 ma. and the surface of the sclera has to be kept dry throughout the entire course of the operation. The length of the application depends on the effect to be obtained. As soon as the sclera shows a grayish and dry area underneath the electrode, the current is switched off, and the electrode is removed. The treatment is repeated on an adjoining area at a distance of a few millimeters. The area to be treated has to be encircled by these applications. After the diathermy applications, several punctures of the sclerochoroid are necessary to permit the escape of the subretinal fluid. This facilitates the apposition of the retina to the choroid. The conjunctival flap is then sutured back to its original position, atropine is instilled, a binocular bandage is applied, and the patient is placed in bed in such a position as to have the vitreous press by gravity on the treated area. This procedure, consisting of the application of spherical electrodes on the surface of the sclera, is called "surface diathermy" by Professor Weve.

The "micropuncture method" consists of piercing the sclera or sclerochoroid with a fine needle electrode in the region corresponding to the retinal tear. The current does not exceed 50 ma., and the length

of time varies between one and three seconds. The depth to which he introduces the needle, the duration of the application and the number of punctures depend on the problem at hand. If the detachment is very much ballooned, the needle is introduced beyond the choroid; if it is of long standing, the application is more lengthy (three seconds); if the tear is large or if there are several tears, the number of punctures is greater, sufficient to surround the area with the tear and a few more additional ones to allow the escape of vitreous. The indications for surface diathermy are: (*a*) a dialysis at the ora serrata and (*b*) a flat detachment. The micropunctures are used in detachments of long standing and in cystlike elevations of the retina, mainly situated further back.

Weve reported the cases of 23 patients treated with surface diathermy. Eleven were cured, and of the remaining 12, some were worse and others remained unchanged; cataracts developed in 4 cases. The vision obtained was between 20/20 and 20/200. Weve's failures were mostly in: (*a*) old patients; (*b*) patients with bilateral detachments; (*c*) those with high myopia; (*d*) those who had undergone previous Gonin operations with scar formation; (*e*) those with small pupils in whom the fundi could not be studied carefully; (*f*) those with uveitis, and (*g*) patients without a tear. He obtained the best results in young people and those who were not myopic. Fifty per cent of the cures were in patients with large tears at the ora serrata. Some of the detachments were from one to several years old.

At the 1932 meeting of the German Ophthalmological Society, Weve communicated on a modification of his micropuncture technic. He is now using a much weaker current (only a few milliamperes) and has discarded the inactive electrode entirely (using only the unipolar electrode). He localizes the position of the tear by the aid of a micropuncture, and checks up its effect on the retina and its location in relation to the tear by immediate ophthalmoscopic examination. Ophthalmoscopy is facilitated by the application of a Zeiss contact glass on the patient's eye during the operation. This prevents drying up of the cornea as well. At this meeting he reported a new series of 7 cases in which he obtained cures; 2 of the patients had previously been operated on unsuccessfully by the Gonin method.

At the same meeting Safar of Professor Meller's clinic (Vienna) reported his experiences with the diathermy treatment of retinal detachments. He leans favorably toward this method and uses a variety of electrodes of his own design. They consist of minute metallic bars with one, two, three or six needle points, from 2 to 3 mm. in length. The latter look like small brushes with a handle that fits into the electrode carrier.

Like the other workers interested in the problem of surgical treatment of retinal detachments, Safar believes that it is essential to produce an inflammatory reaction in the choroid overlying the area of the retinal tear, and that what one is really accomplishing is not occlusion of the tear, but its seclusion. It is important, he says, to cause as little damage to the choroid and retina as possible. This is obtainable by using as small electrodes as one can. After the sclera has been exposed, he drives the needles, or as he calls them "nails," through the sclera by holding them steady with small forceps and pressing them gently against the sclera with the end of the active electrode. He uses about from 50 to 100 ma., a current just sufficient to implant the entire length of the "nails" into the tissue. The little bars with their "nails" are implanted, one next to the other, along a circular line surrounding the tear if it is far from the ora serrata, or in a semicircular line if there is a detachment of the retina at the ora. He does not remove the "nails" before the entire area is thus treated. This prevents the escape of vitreous and collapse of the eyeball. When the procedure is completed, he removes the "nails" and waits for the subretinal fluid to ooze out through the minute openings. He often uses suction by the aid of a small glass syringe provided with a hypodermic needle with a dull tip, applied on the surface of the sclera at each needle entrance. The removal of a sufficient amount of fluid is important. This allows apposition of the retina to the choroid, and subsequent agglutination. Safar often perforates the sclerochoroid with a one needle electrode in several places outside the area of the tear to allow the escape of more subretinal fluid and to produce several foci of choroidal inflammation. For tears situated too far back and difficult of access, he uses the brushlike electrodes, which can be made to penetrate the sclera without injuring the adjoining tissues. His results compare favorably with those of Gonin and Guist, and, in addition, the method does not suffer from the disadvantages already pointed out by others in the course of this review. Safar has seen cures also in cases of detachment with a tear. His method is of too recent date to permit definite conclusions, but it looked interesting and promising to me.

During the discussion of the papers on these newer surgical methods of treatment before the German Ophthalmological Society, Clausen of Halle reported on his own results. He had 37 per cent cures by the Gonin method and 45 per cent cures by the aid of diathermy treatment. Jess of Giessen is also partial to the diathermy method. So far, it can be safely said that the Gonin method has already proved its worth in certain types of cases. This method has been tested out sufficiently and by a large enough number of ophthalmologists to bring out many of its disadvantages and complications. The Guist method seems to

be able to salvage some eyes which are not acceptable to Gonin's operative indications, but, again, it has a great drawback—the length of time necessary for its performance.

Finally, the Weve-Larsson-Safar method presents the advantages of the Guist operation without its shortcomings. It is a matter of time only when the diathermy method will prove its real value. Meanwhile, one should familiarize oneself thoroughly with the technic and principles of the newer ways of treating this disease, which has hitherto been considered hopeless. As matters stand now, there is scarcely an operative procedure in ophthalmology which requires so much preliminary study of the case and so much care, judgment and experience in the performance of the operation as the present-day surgical method of treatment of detachments of the retina. Last summer I had the opportunity of examining a number of patients operated on by Professor Weve and Dr. Safar. I also witnessed a number of operations performed by these two ophthalmologists, and I was impressed by the relative simplicity of their procedures and the results obtained.

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News and Notes

SOCIETY NEWS

Association for Research in Ophthalmology.—The fourth scientific meeting of the association will be held on June 13, 1933, at Marquette University Medical School Auditorium, 561 North Fifteenth Street, Milwaukee. The following program has been arranged: 9:30 a. m. (1) "Chemistry of the Lens," Dr. Cecil S. O'Brien and Dr. P. W. Salit. (2) "Metabolism of the Normal and the Cataractous Lens," Dr. Peter C. Kronfeld. (3) "Etiology of Cataract as Determined by Slit-Lamp Studies," Dr. Arthur J. Bedell. (4) "Mechanism of Senile Cataract," Dr. Daniel B. Kirby. 2:30 p. m. (5) "Possible Influence of Immunological Reactions in Senile Cataract Formation," Dr. Alan C. Woods and Dr. Earl L. Burky. (6) "Study of Cataracta Nigra and Cataracta Brunescens," Dr. Sanford R. Gifford and Dr. Irving S. Puntenny. (7) "Water Equilibrium in Normal and Cataractous Lens," Dr. James E. Lebensohn.

Annual Congress of the Ophthalmological Society of the United Kingdom.—The annual congress was held May 11 to 13, 1933, with Dr. A. H. H. Sinclair presiding. The program was as follows: May 11. (1) "The Present Position of Ultra-Violet Therapy in Eye Disease," Mr. Frank W. Law. (2) "The Value and Technic of the Use of Radon in Certain Intra-Ocular Conditions," Mr. R. Foster Moore. (3) "The Effects of Radium Emanations in Certain Selected Diseases of the Eye; Illustrated by Pathological Specimens," Mr. H. B. Stallard. (4) "Report on a Case of Glioma Retinae Treated by Surface Applications of Radium, with Pathological Findings," Mr. A. D. Griffith. (5) "The Effect of Irradiation of the Eye," Mrs. Phillipa Martin. (6) "On the Projection of the Retinal Quadrants on the Lateral Geniculate Bodies, and the Relationship of the Quadrants to the Optic Radiations," Dr. Ivy MacKenzie, Dr. Spence Meighan and Dr. E. N. Pollock. (7) "The Pathology of a Late Infantile Case of Amaurotic Idiocy, with the Macular Appearances of Tay-Sachs' Disease," Dr. J. G. Greenfield and Dr. S. Nevin. (8) "Cataract in Dystrophia Myotonica," Dr. J. E. Caughey. (9) "A Case of Cataract Extraction in Myotonia Atrophica," Dr. W. Clark Souter. May 12. (1) "Exudative Retinitis in Children," Dr. John Marshall and Dr. I. Chesar Michaelson. (2) "An Ocular Mendelian Puzzle," Prof. Reginald Punnett. (3) Discussion on "Heredity and Eye Disease," opened by Mr. C. H. Usher, followed by Dr. M. Van Duyse. (4) "Two Cases of Recurrent Spasm of the Central Artery of the Retina," Dr. H. M. Traquair. (5) "A Conjunctival Suture Inserted Before Section in Certain Cases of Cataract Extraction," Mr. R. Affleck Greeves. (6) "A Factor in the Etiology of Glaucoma," Mr. W. S. Duke-Elder. May 13. (1) "A Discussion of the Relationship, Direct or Indirect, Between Certain Cases of Herpes Ophthalmicus and Varicella," Mr. L. H. Savin. (2) "Ocular Crystals," Mr. J. H. Doggart. (3) "Relative Ptosis," Mr. E. Wolff. (4) "Report on a Case of Carcinoma of the Lacrimal Sac," Mr. J. N. Tennent.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Aqueous Humor

A COMPARISON OF THE DYE CONTENT OF SERUM ULTRAFILTRATES AND AQUEOUS AFTER INTRAVENOUS INJECTION OF DYES. T. SUZUKI, Arch. f. Augenh. **106:** 458, 1932.

Many experiments have been performed in the past which prove that the concentration of fluorescein in the aqueous is similar to that of an ultrafiltrate of the blood after the dye has been injected intravenously. The amount found in the aqueous is always less than the amount found in whole blood, since a large percentage of the dye is adsorbed by the plasma proteins, and hence is not free to diffuse through the membrane separating aqueous from blood. There is no doubt, however, that the amount of fluorescein which is not so adsorbed diffuses freely across this membrane and is found in the aqueous.

Fischer (*Arch. f. Augenh.* **100-101:** 480, 1929) found a number of dyes which did not come through into the aqueous, although they were found in sufficient quantity in the ultrafiltrate of the blood. He concluded that the electrical charge which these dyes carried could explain their being held back in the blood stream. The present experiments were carried out with a view to confirming this conclusion.

Four acid dyes were injected intravenously, and twenty minutes later the concentration of dye in the blood serum, the ultrafiltrate of the blood and the aqueous was determined. These dyes were of varying molecular size. In every case the amount of dye found in the aqueous corresponded to the amount present in an ultrafiltrate of the blood.

When basic dyes were similarly tested, no traces were found in the aqueous, although large quantities were present in the ultrafiltrate, and the dyes were of small enough molecular size to pass through the membrane.

The author concludes, therefore, that the reason why these basic dyes do not appear in the aqueous is the electrical charge which they carry.

F. H. ADLER.

THE INFLUENCE OF DRUGS ON THE ALBUMIN CONTENT OF THE AQUEOUS HUMOR. A. SASZ, Klin. Monatsbl. f. Augenh. **87:** 543 (Oct.) 1931.

Atropine and scopolamine hydrobromide decrease the content of albumin in the aqueous humor to a small degree, ergotamine to a considerable degree. The albumin content is decreased during the first half hour after the use of epinephrine and increases later. Ephedrine increases the albumin content in the aqueous humor.

K. L. STOLL.

Conjunctiva

A Short History of Ophthalmia During the Egyptian Campaign of 1798-1807. M. Meyerhof, Brit. J. Ophth. **16:** 129 (March) 1932.

The paper opens with a retrospect concerning ophthalmia in Egypt before the French Expedition, and continues with an account of ophthalmia in the French Army, in the Turkish Army and in the British Army and Navy in the campaigns of 1801 and 1807. It closes with the following conclusions:

1. The ophthalmias affecting the French, Turkish and British armies in Egypt during the campaigns from 1798 to 1802 and in 1807 were —as far as the rather vague descriptions left by military surgeons allow them to be identified—the same as those still prevalent in Egypt, that is, a combination of Koch-Weeks', gonorrheal and postgonorrheal conjunctivitis with trachoma. The high percentage of blindness was due to gonococcic infections (which are transmitted, in hot climates, by extragenital means).

2. The contagious nature of the disease, not admitted by French surgeons, was discovered by several young surgeons of the British Army and Navy (Edmondston, Power, Briggs, Reid and Dewar) and established by judicious observations.

3. The improved conditions that have prevailed in the British Army of Occupation in Egypt with regard to ophthalmia, throughout a stay of over half a century, are due to the personal cleanliness of the troops and the high standard of their education in hygiene.

W. Zentmayer.

Four Cases of Parinaud's Conjunctivitis. Longchampt and Reboul, Arch. d'opht. **49:** 88 (Feb.) 1932.

The authors report briefly four cases of Parinaud's conjunctivitis, which were all observed within a month's time. Two cases occurred in two members of the same family who were caring for some sick poultry, one in a farmer in frequent contact with animals that were apparently healthy and the fourth in a midwife who was in the country on a vacation. The ocular involvement was mild. In the first case the edema of the lids and face suggested erysipelas. In the second case the conjunctival lesions would have escaped notice if attention had not been drawn to a swollen preauricular gland. In contrast to the mild local symptoms, there were severe general symptoms, accompanied by a rash similar to that described by Oliver in Mediterranean fever. The authors therefore raise the question as to whether this disease may not be due to a filtrable virus.

S. B. Marlow.

A Lymphomatous Syphilid of the Conjunctiva. Francois, Arch. d'opht. **49:** 91 (Feb.) 1932.

The author describes a case of a conjunctival syphilid in which both the clinical picture and pathologic examination could lead to the confusion of a syphilitic lesion with a tumor of a different nature. Improvement took place under antisyphilitic treatment. The lower fornix of each eye contained a swollen lymphomatous mass, a piece of which was

excised and examined microscopically. Leaving out of consideration primary chancres, syphilitic lesions of the conjunctiva may be divided into four groups: syphilitic conjunctivitis, syphilitic pseudotrachoma, syphilitic scleroconjunctivitis and syphilids of the conjunctiva. The distinguishing features of these are briefly referred to by the author. Syphilids of the conjunctiva are extremely rare. Of all the mucous membranes the conjunctiva is most rarely affected. Three types of syphilid have been described: syphilitic papules, mucous plaques and ulcers. The case reported by the author falls into none of these groups although it belongs to the class of syphilids. The conjunctival localization of syphilis is much more uncommon than other ocular manifestations. The statistics of several authors are cited. S. B. MARLOW.

VERNAL CONJUNCTIVITIS AND SEASONAL VARIATIONS IN EOSINOPHILIA. F. CASINI, Arch. di ottal. **39:** 241 (May) 1932.

The theory which considers vernal conjunctivitis as due to external agents, either bacterial or such as cause anaphylaxis, and that which explains it as due to a constitutional anomaly are compared. If the condition were due to external agents, the eosinophilia of the blood should change with the season, Casini believes, or with the onset of symptoms. In six typical cases blood counts were made from January to June and also after exposure of the body to ultraviolet irradiation. The eosinophilia varied from 7 to 11 per cent in different cases, but was almost constant in each case, showing no influence of season or irradiation. This is considered evidence in favor of a constitutional basis for the disease. In all cases signs of the vagotonic constitution described by Angelucci were present. S. R. GIFFORD.

Comparative Ophthalmology

VARIATIONS IN THE EYES OF TRITON ALPESTRE DURING THE MATING SEASON. A. PEZARD, Compt. rend. Soc. de biol. **110:** 948 (July 19) 1932.

The eye of the gastropod, Triton alpestre, shows an interesting adaptation to a changing environment. During the mating season, this animal, which is otherwise terrestrial and nocturnal, undergoes changes in appearance and becomes diurnal and aquatic. The eyes become larger, and the black irides change to a golden hue. The retina of the mating triton is about one-fifth thinner. The cones are unaffected, but the rods and the other retinal elements are considerably diminished in length. The enlarged eyeball is due to swelling of the vitreous based on increased imbibition. The lacrimal glands during this period are less developed. The shortened rods and the smaller lacrimal glands are transformations adapted respectively to the diurnal and aquatic status.

J. E. LEBENSOHN.

Experimental Pathology

TICK FEVER PRODUCED BY EXPERIMENTAL INTRA-OCULAR INOCULATION. D. COMBIESCO and G. ZOTTA, Compt. rend. Soc. de biol. **110:** 1222 (Aug. 12) 1932.

Some patients with tick fever present ocular lesions—conjunctivitis, keratoconjunctivitis and chemosis—which occasionally precede by

several days the fever and exanthem. This sequence suggests that in these cases the conjunctiva instead of the usual black spot on the skin is the portal of entry. In two volunteers, several drops of an emulsion of infected tick larvae were instilled in the eye. In both a severe conjunctivitis ensued, followed by a mild febrile attack and, in one case, by a typical exanthem.

J. E. LEBENSOHN.

General Diseases

RARE OBSERVATIONS OF SYPHILIS OF THE EYE. E. PUSCARIU, Arch. d'opht. **49:** 37 (Jan.) 1932.

The first case reported by the author is one of Jensen's disease, retinochoroiditis juxtapapillaris. This case differs from those collected from the literature by Friedenwald in that there was no involvement of the iris and ciliary body. Liebreich first described a case in 1863 under the title of syphilitic retinitis. Syphilis and tuberculosis are thought by several authors not to be causative agents in this disease. From the standpoint of the retinal lesion, the author believes that the cases of Mylius, Igersheimer, Liebreich, Scheidemann, Fuchs and Gutmann may be considered in this group. They were all syphilitic, improving under antisyphilitic treatment.

The second case described is that of retrobulbar gumma which was cured by treatment, with restoration of vision. Only five cases are on record, all of them having been diagnosed microscopically after enucleation. The author's case is the only one diagnosed *in vivo,* the clinical picture and subsequent course being consistent with the diagnosis.

S. B. MARLOW.

METASTATIC LESIONS OF EYE IN EXPERIMENTAL SYPHILIS OF RABBITS. A. I. BESSEMANS and J. VAN CANNEYT, Compt. rend. Soc. de biol. **111:**161 (Oct. 3) 1932.

Various organs of the rabbit were inoculated with the same syphilitic strain, and the effect on ocular metastasis was noted. Of 3 rabbits inoculated in the brain, a bilateral corneal lesion developed in 1. Of 247 rabbits inoculated in the testes, ocular metastases developed in 21: cornea, 11; cornea and iris, 8; limbal phlyctenule, 1; syphiloma of the lid, 1. The corneal metastases were bilateral in 14 cases, and unilateral in 5. Mobile spirochetes were present in 8 of 12 corneas examined. Keratitis appeared as a recidive disease in various animals within from six to two hundred and seven days after healing of the primary lesion. The iridic metastases were bilateral in 1 case, and unilateral in 7. Acute and chronic, plastic and nodular forms occurred.

J. E. LEBENSOHN.

EFFECT OF OCULAR TRAUMAS ON THE EVOLUTION OF LOCAL AND METASTATIC LESIONS IN EXPERIMENTAL SYPHILIS. A. I. BESSEMANS and J. VAN CANNEYT, Compt. rend. Soc. de biol. **111:** 238 (Oct. 14) 1932.

In seventy-eight rabbits with active syphilitic lesions, the eyes of which were variously traumatized, a transcorneal puncture in three

favored the localization of metastatic keratitis. In twenty eyes with syphilitic keratitis, corneal scarification or transcorneal puncture did not aggravate the condition. Considering the receptivity of the cornea for the syphilitic virus, the influence of ocular trauma in localizing metastasis is probably insignificant.

J. E. LEBENSOHN.

CLINICAL CONTRIBUTION TO THE STUDY OF OXYCEPHALY. T. BARDANZELLU, Arch. di ottal. **39:** 201 (May) 1932.

The various theories of the causation of oxycephaly are reviewed in detail and a case is reported. A boy of 4 years showed prominent eyes and a marked frontal prominence at birth. The exophthalmos and changes in the skull progressed, and at the age of 1 year, periods of stupor and headache occurred frequently. After this, general development was fairly normal. When seen by the author the skull was narrow, with marked frontal prominence. Vision was: O.D. 1/4; O.S. 1/6. Exophthalmos measured 24 on the right and 26 on the left (Hertel). At spinal puncture the fluid escaped under high pressure. Both disks showed simple atrophy. Roentgenograms showed bony thickening in the region of the anterior fontanel, a concavity in the left frontal region and marked digital impressions. The optic foramina were normal in size. The suture lines could not be seen.

The author accepts as the cause of oxycephaly a premature closure of the sutures, allowing the brain to expand in the direction in which they remain longest open. In this case the frontoparietal sutures closed in their lateral portions, remaining open above, where the compensatory prominence occurred. The left side had evidently closed earliest, accounting for the left-sided concavity. Variations in the location of premature closure account for the various types of skull seen in this and allied conditions.

As the cause of early closure, the author considers most probable a low grade pachymeningitis, indicated here by the history of spasms and the increased pressure of the cerebrospinal fluid. Rickets, although present in his patient, cannot be given causal significance, he believes, since the deformity was present at birth. He is more inclined to agree with Bertolotti that adenoiditis, also present here, could account for the meningitis. A bibliography and photographs are included.

S. R. GIFFORD.

Glaucoma

A SUGGESTION AS TO THE CAUSE OF GLAUCOMA FOLLOWING THROMBOSIS OF THE RETINAL VEIN. D. J. WOOD, Brit. J. Ophth. **16:** 423 (July) 1932.

Four patients studied were under 40 years of age. In two there were many hemorrhages, but glaucoma did not supervene, presumably because of the patients' youth. In two there were few hemorrhages, the vitreous was not stained and glaucoma did not supervene. Five cases were seen in older people. In two the hemorrhages were few, and the vitreous appeared similar to that in the other eye. Glaucoma did not occur. In three there were many hemorrhages, with definite dimness of the fundus

in red-free light. In two cases glaucoma developed, but the patients died before pain demanded excision. In the third case the eye was excised. The suggestion derived from these few cases is that some substance formed from hemoglobin is absorbed into the vitreous, and, if absorbed in sufficient quantity, alters its light-transmitting properties, sometimes causing a turgescence of the vitreous gel.

The author's attention was drawn to the fact that in the fundus of a young woman who had thrombosis with many hemorrhages, though one could see clearly with ordinary electric light, the picture by red-free light was very dim. The vitreous, though clear, would not transmit the blue end of the spectrum.

The author has seen eight other cases of thrombosis of the retinal vein, and whenever the hemorrhages were extensive, the vitreous was found to be more or less opaque to red-free light.

W. ZENTMAYER.

Methods of Examination

HYPERPHORIA AND THE PROLONGED OCCLUSION TEST. C. C. BEISBARTH, Am. J. Ophth. **15:** 1013 (Nov.) 1932.

The prolonged occlusion test was applied in nineteen patients. In the first nine cases, which showed a left hyperphoria before occlusion, the left eye was occluded. In none was the hyperphoria less after occlusion; in all but two cases there was an increase in the hyperphoria after occlusion, which varied in time from one and one-half hours to seven days. In the next seven cases, which showed right hyperphoria, the right eye was occluded. Six showed considerable increase in the right hyperphoria. In only one was the hyperphoria less after the test, a vertical orthophoria being present after four days of occlusion, when one-half degree of right hyperphoria had been present before.

The next eight cases showed no hyperphoria before the test. In five the left eye was occluded; two of these exhibited left hyperphoria of 1½ and 1¾ degrees after comparatively short occlusion. In three cases in which there was no hyperphoria before occlusion, the right eye was occluded; two showed a right hyperphoria after occlusion, and while the degree was not high, the occlusion time was short and the tendency was always toward a hyperphoria of the covered eye.

In each of the last five cases a right hyperphoria was present before occlusion. The left eye was occluded. In one a right hyperphoria of three-fourths degree was replaced by a left hyperpohria of 2½ degrees. In one a right hyperphoria of 1 degree became a left hyperphoria of three-fourths degree. In the last three a right hyperphoria was less after the left eye was covered.

From these results it seems that the use of the test to estimate the amount of hyperphoria to be corrected prismatically would lead to error. Few users of the test have been willing to prescribe the full correction found after occlusion, and this may account for the good results and satisfaction with the test reported by many authors.

W. ZENTMAYER.

Ocular Muscles

EYE MOVEMENTS IN THE NEW-BORN. BARTELS, Deutsche med. Wchnschr. **58:** 1477 (Sept. 16) 1932.

Two reflexes found in the baby at birth are described. The first is demonstrable particularly in the premature infant and at times in the normal, sleeping new-born and is called by the author "doll eye movement." If the head is moved in one direction, the eyes deviate in the opposite direction without the usual quick return component. This is found especially in the premature infant in whom at times the returning component is found to be very slow, while in the normal infant the quick component of the so-called turning nystagmus is very marked.

A lid reflex described by Bárány and others is found during the first two months of life and then disappears. If the infant's eyes are closed while the child is lying on its back, then by a passive movement of the head forward the eyes are caused to open. Another reflex found first by Lyon in fish and later by Bárány in infants, called the neck-eye movement, disappears after the first week of life. If the body is flexed while the head is in a fixed position, the eyes are often seen to move in nystagmoid fashion. The author does not agree with Catel that the origin is the same as that found in miner's nystagmus, but rather favors the view that it is an optical type such as is shown by the blind. (Bibliography.)

L. L. MAYER.

Pharmacology

EXPERIENCES WITH "PANTOCAINE" (*p*-BUTYLAMINOBENZOYL *di*-METHYL-AMINOETHANOL HYDROCHLORIDE). K. SCHMIDT, Klin. Monatsbl. f. Augenh. **87:** 821 (Dec.) 1931.

The author used a 1 per cent solution of this drug in about 1,200 operations, comprising removal of foreign bodies, cauterization of the cornea and punctures of the anterior chamber, and in tonometry. He obtained prompt anesthesia which lasted as long as was required. No corneal lesions resulted. Schmidt considers the preparation decidedly less poisonous than cocaine. Bartels, Hessberg and others have corroborated Schmidt's view. Hessberg uses the drug in solutions of from 0.5 to 1 per cent with good results; he never saw corneal lesions result but had occasional patients who complained of a burning sensation, especially after the use of the ointment.

K. L. STOLL.

Physiology

INFLUENCE OF THE SYMPATHETICS ON MUSCLE GLYCOGEN. HERRIN and MEEK, Am. J. Physiol. **97:** 57, 1932.

The sympathetic chain was removed on one side in a series of rabbits and dogs. The denervation caused a diminution in the glycogen content of the muscles involved. This was most marked in the tonic muscles of the respective species.

There is ample proof that muscle tonus is not directly maintained by the sympathetic nervous system, but it may be supported indirectly by the glycogenic activity of the sympathetic nerve fibers in aiding the postural muscles to maintain their supply of available energy. These studies indicate that sympathetic impulses cause a deposition of glycogen

in the muscles. This seems to be independent of any changes in the circulation, as an increased flow of blood to a limb does not of itself cause any decrease in muscle glycogen.

F. H. ADLER.

PASSAGE OF FLUID THROUGH THE HUMAN CAPILLARY WALL DURING VENOUS CONGESTION. E. LANDIS, L. JONAS, ET AL., J. Clin. Investigation **11:** 717 (July) 1932.

The passage of fluid through the capillaries in the arm was calculated from the changes in cell count, hematocrit values and percentage of protein found in the blood before and after the application of venous stasis. A cuff was placed on the arm and inflated to a known pressure. Samples of blood were withdrawn before the application of the cuff and after the cuff had been applied for thirty minutes.

The authors find that a small rise in venous pressure is sufficient to cause a definite loss of fluid from the circulation into the tissues. This fluid contains increasing amounts of protein as the venous pressure is raised. When the venous pressure is at 80 mm. of mercury the protein content is as high as 1.3 per cent. An analysis of the protein in edema fluid showed a very low percentage, i. e., 0.3 per cent.

F. H. ADLER.

THE PART PLAYED BY THE SYMPATHETIC SYSTEM AS AN AFFERENT MECHANISM IN THE REGION OF THE TRIGEMINUS. HARRY HELSON, Brain **55:** 114, 1932.

The author examined a number of patients at various intervals following subtotal section of the trigeminal root (operation by Dr. Charles H. Frazier) in order to determine to what extent sensation remained in the face after deprivation of the trigeminal supply and also to determine if any restitution in sensory function took place as time went on.

Six forms of sensation were studied, i. e., light and deep pressure, cold, heat, pain, two-point pressure limen and the ability to localize a spot within the affected area. It was found that after operation the superficial pressure was entirely lost when tested by punctate pressure, except in certain isolated spots which occasionally were hyperalgesic. There was never any restitution in the sense of light pressure. Up to one month after operation deep pressures were not felt unless the stimulus was very heavy. After this time, however, restitution of function took place and increased so that after four years the sensitivity to deep pressure had increased at least a hundred fold. The sensation of heat as such was lost after the operation, but when the sympathetic supply of the face was intact the patients would complain of a stinging or pricking sensation. When the sympathetic supply was eliminated (in three patients a thoracic sympathectomy was performed), all sensation of heat stimuli disappeared. The sensation of cold was entirely lost.

The sensation of pain was entirely lost and never returned, with the exception of the touch spots which reacted as already described. After operation the ability to localize the spot was lost at first, but recovery of this sense gradually took place as though some other nerve was "learning" the function formerly mediated by the trigeminus. The two-point limen function was irretrievably lost.

These results indicate that the facial fibers may be responsible for most of the sensation when the trigeminus is lost but that in the case of heat stimuli at least the sympathetic supply is chiefly concerned. It is postulated, therefore, that the sympathetic has an afferent as well as an efferent function. This assumption, the author believes, places one in a better position to understand the sensations which remain and develop after section of the sensory root of the trigeminus.

F. H. ADLER.

PRESSURE AND CIRCULATION IN THE HUMAN EYE. R. SONDERMANN, Arch. f. Augenh. **106:** 319, 1932.

As a result of previous work, the author concludes that the intra-ocular pressure is maintained by the volume of blood in the intra-ocular capillaries. This is made possible by the constriction of the veins as they course through the sclera during embryonic life. This constriction creates a turgescence in the choroidal capillaries, raising their pressure and hence intra-ocular pressure. The pressure in the uveal set of vessels is much higher than that in the retinal system.

The present voluminous article presents evidence of an embryologic nature that the origin of the intra-ocular blood vessels and the venous drainage from Schlemm's canal is such as to satisfy the demands made by the author's previous theories.

He believes that Schlemm's canal originally consists of a group of sixty or seventy radial coursing veins, forming a circumscribed dilatation and joined together by numerous capillary connections. In the course of about three months the veins lose their function in human beings and monkeys, and transport aqueous humor instead of blood. In the lower animals the canal still contains blood. A schematic drawing of the relations of this canal to the anterior ciliary veins into which it opens is offered to explain how aqueous is aspirated from the anterior chamber into the veins.

In order to prove the nature of this mechanism of exit of fluid from the anterior chamber the author has experimented on Macacus monkeys in the following manner: A fine knife needle is inserted in the anterior chamber through the periphery of the cornea, causing a small hemorrhage in the chamber. The slight fall in intra-ocular pressure occasioned by the entrance of the needle and loss of aqueous is soon recovered from; four hours later the eye is enucleated, and histologic sections are prepared. As a result of these studies he believes that the aqueous humor itself drains entirely out of the eye through Schlemm's canal, but that corpuscular elements, such as red blood cells, find their exit from the eye by way of the iris and ciliary body. F. H. ADLER.

THE SO-CALLED CORNEAL AND SCLERAL REFLEXES. W. S. SURAT, Klin. Monatsbl. f. Augenh. **87:** 645 (Nov.) 1931.

It is comparatively easy to demonstrate the preservation of the central reflex arc which conducts the corneal and conjunctival reflexes. The author undertook a series of tests with the view of localizing this reflex arc. In five hundred school children and one hundred healthy adults the corneal reflexes were found to be constant, but no reflex could be obtained by irritiation of the episcleral conjunctiva. Closure of the lids

on coarse wiping of the conjunctiva, causing epiphora, is considered to be a voluntary defensive reaction if sensitiveness is intact. Even though the corneal reflex is vivid in new-born children, no reflex was observed on the approach of an object. The corneal reflex became less active during infancy. The absence of reflex disturbances in Parkinson's disease, the slight disorder in hemiplegias and the total absence of the reflex in diseases of the trifacial-facial peripheral reflex arc suggest the presence of a brain stem reflex arc; this is supplemented by the sensomotor portion of the cerebral cortex. The closure of the reflex arc in the subcortical ganglions, looked for by many authors, must be denied.

K. L. STOLL.

Refraction and Accommodation

CONTACT GLASSES. B. W. RYCROFT, Brit. J. Ophth. **16:** 461 (Aug.) 1932.

This paper covers a brief historical record of contact glasses, their physical, physiologic, optical and psychologic properties.

The disadvantages of such glasses are: An easy method of insertion, suitable to all patients, has not yet been found; discomfort is invariably experienced at first, and may lead to their discontinuance by the patient; they are costly (about 4 pounds each), and they are a potential source of danger to the eye, through injury, though no such case has yet been recorded. There is no agreement as to whether a tight-fitting lens or one that gives a slight laxity of correspondence in movements between the eye and the glass is more comfortable. The author believes that the discomfort arises from lid pressure and partly from a slight amount of movement in the glass, which allows the shoulder to ride onto the corneal surface. Precautions must be taken to avoid too small a corneal radius and to use phenacaine liberally.

The best indications for the use of contact glasses are: (1) cases of advanced conical cornea in which correction with spectacles offers no improvement and in which there is no great opacity of the corneal apex, (2) cases of old corneal irregular ulceration in which the ensuing scars have no great density, (3) cases of neuroparalyitic keratitis in which corneal anesthesia has hitherto called for a tarsorrhaphy to prevent external injury and (4) selected cases of higher refractive errors.

The author has devised a simple apparatus for the insertion and removal of such glasses.

A case is reported of conical cornea in which visual acuity was improved in the right eye from 1/60 to 6/7.5 and in the left eye from counting fingers to 6/7.5 (partial). The case has been under observation for nine months and at present the glasses can be worn with comfort for eight hours.

W. ZENTMAYER.

A STATISTICAL STUDY OF THE FREQUENCY OF MYOPIA. M. ROHNER, Schweiz. med. Wchnschr. **62:** 706 (July 30) 1932.

This article received the prize of the medical faculty of Zurich. The statistics cover examinations of 11,251 recruits from five cantons and the city of Zurich, Switzerland, for the year 1924-1925. The author cites the fact that because the examinations were not made under

cycloplegia, were entirely subjective and were only partially made by ophthalmologists, the figures are not entirely reliable. The patients were about 19 or 20 years of age, and were grouped both as to occupation and degree of refractive error. The myopic subjects were classified in four groups, according to the degree of myopia: up to 3, from 3 to 6, from 6 to 9 and more than 9 diopters. Of the 11,251 persons, 672, or 6 per cent, were myopic; 3.3 per cent had myopia of more than 6 diopters. Comparison of the city and country bred revealed a higher percentage of persons with myopia among the former, 8.6 per cent as compared with 3.6 per cent. Students, teachers, tailors, watchmakers and mechanics doing finer degrees of close work showed from three to four times the percentage of myopia found in farmers and persons of similar occupation. The author concludes that the frequency of myopia is characteristic of the more cultured group. A most complete bibliography and several interesting tables of classifications accompany the article.

L. L. MAYER.

Retina and Optic Nerve

RECURRENT INTRAOCULAR HEMORRHAGE IN YOUNG ADULTS (EALE'S DISEASE). H. P. HUTCHINSON, Brit. J. Ophth. **16:** 513 (Sept.) 1932.

The paper includes an account of the disease, a summary of the literature, a report of five cases and some speculation as to the etiology.

It is pointed out that von Graefe recognized this group of cases twenty-four years before Eale's publication, as in 1855 he mentioned the disease and described periodic intra-ocular hemorrhages in young persons who had often had previous epistaxis.

The chief matter of interest in Eale's disease is its etiology, which is still unknown. Eale's own theory as to the importance of constipation and intestinal disturbance as a starting point does not seem to have received much support. A deficiency in blood calcium, prolonged bleeding time and delayed coagulation of the blood seem to be ruled out in almost every case in which these points have been investigated. Zentmayer's theory that there might be a disturbance in the activity of the ductless glands does not seem to have been tested sufficiently in actual practice. The fact that thyroid extract benefits some patients gives some support to the theory. It would be interesting to try the effect on the disease of injections of epinephrine or of ephedrine given by mouth.

The theory that tuberculosis is a factor lacks any concrete evidence in its support. Krauss has suggested a condition of weakness or local disease in the choroidal or retinal vessels permitting rupture when other conditions are favorable. Why then does the condition start so abruptly in adolescence, only to cease, also abruptly, after several months or years? If an abnormality of the blood, why does it affect only the eye and nose? Is it because there is lack of counterpoising support in these situations or do hemorrhages occur, unrecognized, in other parts of the body? Another possibility is a vitamin deficiency allied to scurvy.

The disease is probably due to a deficiency in some blood constituent or to the presence of some toxic product, causing damage to the endothelium of the smaller branches of the retinal vessels with resultant diapedesis. The occurrence of hemorrhages chiefly in the eye may be due to a lack of supporting connective tissue around the retinal vessels.

W. ZENTMAYER.

SURGICAL ELECTROCOAGULATION IN DETACHMENT OF THE RETINA: EXPERIMENTAL PRODUCTION OF HOLES IN THE RETINA WITH SUBSEQUENT RETINAL DETACHMENT. R. SALUS, Klin. Monatsbl. f. Augenh. **87:** 752 (Dec.) 1931.

Tempted by some apparent advantages of surgical diathermy over thermocautery, Salus attempted to obtain the reattachment of the retina by single or repeated insertions of a diathermic needle through the sclera into the eyes of rabbits. Operations on three hopeless human eyes followed, under special precautions. Therapeutic results were obtained in none of these cases. The result was so discouraging (hemorrhages occurred into the vitreous and holes formed in the retina) that Salus will not use electrocoagulation again in detachment of the retina.

On the other hand, the experiments showed that detachment of the retina can be produced by experimental production of holes in the retina. According to these experiments, it seems that the holes are actually the primary cause of the detachment.

K. L. STOLL.

BASILAR MENINGITIS FOLLOWING TUBERCULOSIS OF THE OPTIC NERVE: REPORT OF A CASE. G. VON SZABO, Klin. Monatsbl. f. Augenh. **87:** 805 (Dec.) 1931.

A case of primary tuberculous infection of the optic nerve giving rise to tuberculous basilar meningitis is reported. A girl, aged 6 years, had noticed failing vision in the left eye, without pain or inflammation, for four weeks prior to her admission to the hospital. At the hospital a diagnosis of basilar meningitis of a tuberculous nature was made, and edema of the left disk with retinal hemorrhages and yellowish-gray discolorations of the retina was observed. The patient died eight days later, presenting meningeal symptoms. Histologic examination of the globe showed the retina to be thickened by edema to twice its size, the disk was prominent, edematous, larger hemorrhages were seen in the deeper fibers, and migrating cells were present in the intravaginal spaces. Four millimeters behind the lamina cribrosa the central vessels were surrounded for a distance of 7 mm. by tuberculous granulations, containing epithelioid, round and giant cells. Here the nerve fibers were completely destroyed. The focus in the optic nerve was the primary lesion, as the meningeal symptoms developed over three weeks after the beginning failure of the vision. Similar cases of an ascending meningitis, arising from the eye, are quoted.

K. L. STOLL.

Trachoma

PROPHYLAXIS OF PANDEMIC TRACHOMA. TALBOT, Rev. internat. du trachome **9:** 127 (July) 1932.

In endemic and pandemic regions, trachoma dates from earliest infancy, and the acute invasions seen in later years are but superinfections. The spread of trachoma requires intimate and continuous contact with the infecting agent. An antitrachomatous campaign must be mainly concerned, therefore, with school children, women and infants. Visiting nurses are essential.

J. E. LEBENSOHN.

TREATMENT OF TRACHOMA. K. V. MORMOLEVSKY, Sov. vest. ophth. 1: 156 (April) 1932.

The author treated thirty-two patients with various stages of trachoma by injections of a preparation having the following formula:

	Gm. or Cc.
Sodium chloride	0.30
Sodium sulphate	0.25
Calcium glycerophosphate	0.50
Sodium nucleinate	0.50
Phenol	1.0
Distilled water	100.0

From 0.5 to 1.0 cc. of this solution was injected subconjunctivally into the fornices of both lids every two days, until from fifteen to twenty injections were given. Drops of the same solution were instilled in the intervals between the injections. There was a marked improvement in thirteen patients with the first stage of trachoma; the follicles began to "melt," and, at the end of the treatment, had absorbed. In all patients with the second stage of the disease there was only a subjective improvement, with a slight clearing of the corneal opacities. No complete cure was observed in any of these cases. No improvement was noticed in the third stage of trachoma. The author recommends this method of treatment only as supplementary to the usual treatment.

O. SITCHEVSKA.

USE OF RADIUM THERAPY IN TRACHOMA. A. N. KRUGLOFF and G. S. LIORBER, Sov. vest. ophth. 1: 196 (May) 1932.

The authors studied the action of various doses of radium on trachoma, and the advantages of this treatment as compared with others. The physicochemical character of radium, its emanations as the result of disintegration and the dosage are given in detail.

The authors have used radium treatment in twenty-five trachomatous patients of various ages since 1929. Of these, thirteen suffered from trachoma in the first stage and twelve from trachoma in the second stage. The more severely affected eye was treated by irradiation, while the usual treatment was applied to the other eye. The period of observation was from one to eighteen months. A filter of radium (from 0.1 to 1 mm. of copper in a flat rubber case) is held on the closed lid by adhesive plaster. The usual dosage was 8 mg. hours per period of treatment, which varied from one to four hours. The treatment was repeated from one to six weeks, corresponding to the length of time of each treatment. In those cases in which small doses were not sufficient, the combination with large doses was more effective. The treatment is painless, so that it can be employed without hospitalization.

According to the results of radium therapy, the patients were divided into three groups. Very good results were obtained in the first group of eight patients. In three of these, the follicles absorbed entirely without leaving cicatrices. A pathologic examination of the conjunctiva of one of the patients, however, showed the presence of dense infiltration with lymphocytes. Fair improvement was evident in the second group

of eleven patients, and six patients of the third group showed no improvement. The decrease of the follicles could be noticed in from ten to twenty days after the first treatment. The disappearance of the follicles was observed in from three to four months after the first treatment. Roentgen therapy was also employed by the authors in ten patients with the first and second stages of trachoma.

The authors draw the following conclusions: 1. Radium therapy gives good results in trachoma in the first and second stages. 2. Radium treatment in trachoma with pannus may cause an exacerbation of the corneal process. 3. The method is painless and can therefore be used in the dispensary. 4. The action of radium and roentgen rays is analogous, but radium therapy is preferable because it effects deep infiltration readily. 5. Radium therapy does not yield permanent results and therefore cannot be recommended as a method in itself. 6. This is a preliminary report, and the study will be continued.

O. SITCHEVSKA.

Tumors

GLIOMA OF THE OPTIC NERVE. B. GLUCK, Brit. J. Ophth. **16:** 406 (July) 1932.

A girl had the left eye removed seven years before the present observations. There had been a history of injury followed by increasing proptosis. The eye was blind and the optic disk pale. A mass was present at the apex of the orbit. Radium was inserted into the orbit. This was followed by an increase in the proptosis, ulceration and perforation of the cornea. The eye was enucleated. Later radium was again applied. When seen by the author the lids were completely adherent to each other. The orbital contents were tense and fluctuating. The cyst was aspirated, and iodized poppy seed oil 40 per cent was injected. A roentgenogram showed the iodized oil above the roof of the orbit and also behind the apex, in addition to masses in the orbital cavity. Death occurred about three weeks later. The roof of the orbit was absent, the atrophy of the bone extending up to the superciliary ridge. A cystic growth in the lower part of the left frontal lobe extended down to fill the orbit, pushing the dura before it. The left lateral ventricle was greatly enlarged, the right one less so. The tumor extended up to involve the greater part of the lenticular nucleus. The tumor of the brain showed the characteristic structure of a glioma. The stump of the optic nerve was recognized in the extradural tissue found at the apex of the orbit; astrocyte cells were present within the dural sheath, but on the whole the tissue had more the character of neural granulation tissue than of true neoplasm.

The case is considered to have additional interest because of its being the first recorded, so far as the author could trace, in which radium therapy was used as the primary and only treatment—i. e., without previous removal of the tumor, or as much of it as was accessible for removal—and because of the diagnostic problem presented by the case before the previous history could be traced.

The author discusses the various aspects of primary tumors of the optic nerve. The article is illustrated.

W. ZENTMAYER.

MELANOCARCINOMA OF THE EYE AND ORBIT ARISING FROM THE PIGMENT EPITHELIUM: REPORT OF A CASE. G. BIETTI, Klin. Monatsbl. f. Augenh. **87:** 459 (Oct.) 1931.

The author renders a detailed description of the pigmented tumors of the globe, and reviews the literature. He reports the case of a man, aged 63, with a perforating melanocarcinoma of the choroid. Exenteration of the orbit was done, and simultaneously the orbital portion of the maxillary bone was removed. The nasal adnexa were found intact. The patient died of metastases to the liver and the spinal cord and regional recurrences. An exact histologic report, illustrated with many photomicrographs, furnished proof of the existence of a melanocarcinoma, originating not alone from the ciliary body but also from the entire pigment epithelium, including also the divers strata of the epithelium of the retina.

K. L. STOLL.

Uvea

ELECTIVE LOCALIZATION IN DETERMINING THE ETIOLOGY OF CHRONIC UVEITIS. E. C. ROSENOW and A. C. NICKEL, Am. J. Ophth. **15:** 1 (Jan.) 1932.

Previous experimental work is referred to in which streptococci and pneumococci appeared to have acquired specific or elective affinity for certain tissues or organs. When these organisms were derived from patients with an acute condition, the resulting ocular reaction was more marked and more progressive than when they were obtained from chronic ocular diseases. The authors describe different methods of inoculating animals with streptococci. The material for this study was obtained from ten patients with a diagnosis of hyalitis with vitreous opacities in two, iritis in three, iridocyclitis in two, follicular corneal ulcers in one, vitreous opacity with hemorrhage in one and conjunctivitis in one. Forty-five rabbits and eight dogs were used. Of the former, 88 per cent revealed a temporary ocular reaction in varying degrees. The teeth of seven dogs were infected with four strains derived from patients with iritis. These strains had caused typical reactions in the eyes of rabbits and one dog following intravenous injection. In no instance was iritis or uveitis associated with clouding of the aqueous humor observed in the course of from four months to three years. This was true despite the fact that the streptococcus isolated many months later from the infected teeth of three of the dogs had retained specific affinity for the uveal tract, as determined in rabbits on intravenous injection. The ocular reactions from killed cultures and filtrates are also described. The strains of streptococci possessing specific affinity for the eye were found to be very much alike. Four illustrative cases are described, two of uveitis, one of choroiditis and one of marginal keratitis.

W. S. REESE.

Society Transactions

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

ALEXANDER G. FEWELL, M.D., *Clerk*

April 21, 1932

LEIGHTON F. APPLEMAN, M.D., *Chairman*

EXHIBIT OF SPECIMENS. DR. BURTON CHANCE.

Dr. Burton Chance exhibited the following:

1. A rather large dermoid cyst containing rolls of densely black hair that had been dissected from the orbit of a girl of 17. The tumor had become increasingly prominent in the past year, and the bluishness of the eyelid quite distinct. The approach was through the eyelid. The cyst was distended by a thick oleaginous fluid.

2. Three lenses extracted in their capsules, each showing traumatic cataract caused by penetration and lodgment of a foreign body.

3. Bilateral vascular new growths at each limbus, covered by the upper lids, and suggesting sarcoma or angioma of the conjunctiva, removed from an old Italian woman. Sufficient time had not elapsed for histologic study.

WOUND OF THE GLOBE BY EXPLOSION OF A SHATTER-PROOF WINDSHIELD. DR. H. MAXWELL LANGDON.

To melt ice frozen on the windshield, the patient placed a candle on the ledge inside the shatter-proof glass. An explosion shortly occurred, making a hole about 2 inches (5 cm.) in diameter on the inner plate, and one about one third this size in the outer plate. At the time of the explosion, the driver felt a blow on his right eye, and hemorrhage occurred from a penetrating wound of the sclera near the upper limbus. The wound was in median line, about 3 mm. long, extending upward from the corneoscleral junction, with a small bead of iris protruding. There had been a small hemorrhage into the vitreous which had rendered it somewhat cloudy, but which did not prevent a view of the uninjured fundus. The protruding iris was excised, and the wound healed promptly, with vision normal.

Heat may be applied with impunity to ordinary glass, but heat applied to shatter-proof glass generates from the cellulose partition a gas of great expansive force.

MELANOTIC SARCOMA OF THE CHOROID OCCURRING AT THE OPTIC DISK IN AN EYE WITH A PREVIOUS DETACHMENT OF THE RETINA. DR. H. MAXWELL LANGDON.

A man, first seen in October, 1929, stated that the vision of the right eye had been blurred for two years. A detachment of the retina occupied the whole lower portion of the fundus, with nothing to suggest an intra-ocular growth. Transillumination over the detached area showed no interference with transmitted light.

In February, 1932, the patient again sought advice, because of a totally blind and intensely painful right eye. The right pupil was 5 mm. in diameter, with no response to external stimuli; the media were too hazy to permit a view of the fundus; the tension was 47. Two days later the right eye was enucleated.

Pathologic examination showed melanotic sarcoma at the optic disk. No new tissue was found in any other portion of the globe. It is not unusual to find in a case of retinal detachment with a secondary rise in tension that an intra-ocular growth has been the starting point of the whole process. In this case there was an eye in which a neoplasm developed apparently after a simple detachment of the retina.

MICROPHTHALMOS WITH COLOBOMA OF THE IRIS, CHOROID AND DISK. DR. HUNTER W. SCARLETT.

Miss A. W., aged 31, presents a microphthalmic right eye with a coloboma of the iris, choroid and disk, and also a small congenital cataract. The left eye shows a coloboma of the iris and choroid. Vision in the right eye was limited to perception of hand movements. Vision in the left eye was 6/30, corrected to 6/10.

The fetal ocular cleft normally closes early in fetal life. If there is a defective closure in the extreme posterior part of the cleft, there is a coloboma of the sheath of the optic nerve; if the defective closure is in the intermediate part of the cleft, a coloboma of the choroid results, or if it is near the anterior lip of the cup, there is a coloboma of the ciliary body and the iris. A defect along the entire length of the cleft, as is indicated in the right eye, presents a coloboma of all the structures mentioned.

LEBER'S DISEASE. DR. T. B. HOLLOWAY.

This article appeared in full in the May issue of the ARCHIVES, p, 789.

DISCUSSION

DR. WILLIAM ZENTMAYER: Soon after Mr. Fisher advanced his theory as to the possible etiology of Leber's disease, I reported the history of two families in which two male members of each family were affected. As in Dr. Holloway's patient, in one of my patients the scotoma had broken through above, and it is probable that a lesion of the pituitary gland would have been considered had it not been for the family history. Roentgenograms were made of all four patients by Dr. Pancoast, and all showed either a high normal or an enlarged sella.

As a control, Dr. Pancoast made a roentgenogram of the pituitary region of the sister of one of the patients, and found that the sella

was enlarged. The eyes were then examined, and small relative central scotomas were found.

It is difficult to reconcile recovery of function in a condition considered to be one of abiotrophy, as by the term "abiotrophy" is meant a condition in which the cells at birth have only vitality enough to exist until they have their full development; at that time they begin to undergo degeneration.

As pigmentary degeneration of the retina occurs more frequently in the male than in the female, it has been suggested by Wibaut that the administration of a female hormone might supply a deficiency in those males affected, and good results have been claimed from its use. As Leber's disease shows the same sex predominance, a similar therapeutic suggestion can be made.

DR. H. MAXWELL LANGDON: Two men for whom I have the utmost respect have put forth an explanation for the occurrence of Leber's disease, and they have ascribed it to abiotrophy. I refer to Sir William Gowers and Mr. Treacher Collins. Abiotrophy, or congenital deficiency of the vitality of the tissues, signifies a degenerative process from the onset, with steady development of the atrophy until the condition terminates in complete loss of function. One of the best examples of this process in ophthalmology is, I think, pigmented degeneration of the retina, when there is a degenerative, noninflammatory, steady process from the onset.

If Leber's disease is abiotrophy, in other words, a degenerative process, I cannot understand how there can be a case in which the condition can progress steadily toward visual loss as in Dr. Holloway's case, and then such remarkable improvement occur, with restoration of function. It seems to me that abiotrophy will not explain the conditions which have existed in his case, and one must look elsewhere for a satisfactory reason for the course of events.

DR. BURTON CHANCE: I have seen five members of the family from which Dr. Shannon presented two sons exhibiting signs of degenerative disease of the optic nerves. I found three members affected, but could not elicit any information concerning the probability of there being other cases in the family.

I find it difficult to place these cases among the hereditary atrophies, as there is not a clear connection with a previous generation. Certainly the conditions are essentially atrophic, and this atrophy I consider as being dependent on some peculiarity of nutrition in each of those affected, rather than as exemplifying the effects of an inherited tendency.

DR. WILLIAM C. POSEY: I saw three generations of a family with Leber's disease. The primary case was in a young man, aged about 20 years; his uncle and his grand-uncle were also affected.

In searching for a cause, I looked into the literature and was impressed by a theory advanced by Berger, of Paris, that Leber's disease was probably caused by an anomalous condition of the sphenoid bone, the optic nerve being affected in its passage through a too narrow optic foramen. All three patients I saw had very flattened occiputs, and it seemed as though the condition might have been due to anomalies in the sphenoid bone.

DR. CHARLES E. SHANNON: The diagnosis of Leber's disease in these two cases may seem doubtful, as no evidence of this disease could be traced in the parents, the grandparents or the great-grandparents. On the other hand, may we not conceive it possible that this condition may have originated in this generation?

Furthermore, the history of the cases, the appearance of the disks and the typical fields, together with the results of the physical and laboratory tests, seem to point indisputably to hereditary atrophy of the optic nerve.

Therapy in Leber's disease is of doubtful value. The suggestion of diathermy was made by Dr. Schmidt, who referred the patients to me, and I saw no reason why it should not be tried. In addition, preparations of the anterior lobe of the pituitary gland, high frequency current and strychnine have been employed. The improvement in vision is encouraging, although it may be merely a remission, which sometimes occurs in these cases.

SYPHILIS IN THE THIRD GENERATION. DR. JOSEPH V. KLAUDER.

Syphilis in the third generation is rare; few cases have been reported. Two children from the Wills Hospital Clinic for the treatment of ocular syphilis were presented.

CASE 1.—A girl, aged 12, the oldest of four children, when first seen, had typical interstitial keratitis of both eyes. The second eye became involved one month after the first eye. The results of the clinical examination were essentially negative except that the patient was overweight and had infected tonsils. There were no stigmas of congenital syphilis. The Wassermann reaction was four plus; the luetin test was positive. Clinical examination of two younger brothers and one younger sister gave negative results, and the Wassermann reaction of each was negative. The Wassermann reaction of the mother was negative, and there had been no miscarriages. The father denied ever having had venereal disease. His clinical examination gave essentially negative results, and his Wassermann reaction was four plus. The father had seven brothers and sisters, all living; one had a hemiplegia at the age of 31. His mother died of a cause unknown to him. His father (the patient's grandfather) had tabes and is now dead.

CASE 2.—A girl, aged 19, an only child, when first seen showed typical active interstitial keratitis, but presented no other stigmas of congenital syphilis. Her Wassermann reaction was four plus. She is married, has had one miscarriage, and one child, aged 16 months. This child is well nourished. The clinical examination gave essentially negative results, the Wassermann reaction of the blood was four plus, and the luetin test was positive. The patient's mother (baby's grandmother) had one miscarriage and no stillbirths. She gave a definite history of having been infected with syphilis soon after marriage, of having had a secondary eruption, of positive Wassermann reactions, and of having had antisyphilitic treatment. The Wassermann reaction of the blood was four plus. Her husband died of cancer. The hospital where he died informed me that his Wassermann reaction was negative.

In the first case, although the child's grandfather had tabes, acquired syphilis in the father cannot be definitely excluded, hence syphilis in the

third generation is not certain. In the second case, however, syphilis in the third generation is more likely, since the child's mother had interstitial keratitis, and the grandmother gave a definite history of infection with a Wassermann reaction of four plus.

ALEXANDER G. FEWELL, M.D., *Clerk*

Oct. 20, 1932

LEIGHTON F. APPLEMAN, M.D., *Chairman*

RETAINED PUPILLARY MEMBRANES. DR. MARY BUCHANAN.

The patients, twin girls aged 9 years, who were born three weeks prematurely, came to the clinic for refraction. On examination, both of them showed retained pupillary membranes, in one at 8 o'clock, and in the other between 10 and 11 o'clock. The vision was 6/9, corrected to 6/6, in each case, and the fundi were normal.

PULSATING EXOPHTHALMOS FOLLOWING TRAUMA. DR. L. F. APPLEMAN.

Pulsating exophthalmos developed following trauma by a fist. Three days after trauma there were diplopia, complete paralysis of the oculomotor and sixth nerves and complete ptosis. A distinct bruit persisted over the temporal region, and a thrill was felt on palpation over the globe. Ophthalmic examination showed very tortuous retinal veins but no hemorrhages. There was no optic neuritis. Examination of the spinal fluid showed abundant red corpuscles, and a negative Wassermann reaction. The roentgenogram revealed no fracture. Ligation of one or more branches of the carotid artery would seem to be indicated.

AMAUROSIS AFTER UTERINE HEMORRHAGE WITH RESTORATION OF VISION FOLLOWING TRANSFUSION. DR. H. MAXWELL LANGDON and DR. GEORGE M. LAWS.

This paper will be published in full in a later issue of the ARCHIVES.

DR. BURTON ALEXANDER RANDALL AS AN OPHTHALMOLOGIST. DR. BURTON CHANCE.

To the present generation of practitioners the late Dr. Randall was known as an otologist, yet, in his early professional days, he was distinguished among ophthalmologists. His greatest contribution to ophthalmology concerned refraction and its problems. Randall employed roentgenography and ophthalmometry as well as the subjective methods. Randall joined others who urged that the eyes of school children and adolescents should be systematically protected by properly adjusted spectacle lenses. Randall lived to see the world's recognition of America's distinct contribution to the science, and today the principles which he so strongly advocated are inherent in the fabric of ophthalmology.

ROYAL SOCIETY OF MEDICINE, LONDON, SECTION OF OPHTHALMOLOGY

Jan. 13, 1933

A. C. HUDSON, F.R.C.S., *President, in the Chair*

A CASE OF BLOOD STAINING OF THE CORNEA. MR. A. C. HUDSON.

As a rule, in cases of blood staining of the cornea there has been glaucoma, with no wound or spontaneous hemorrhage. Following an injury thirteen months ago, the eye showed increased tension for about ten days, followed by blood staining of the cornea. The opacity cleared up from the periphery toward the center, simulating in appearance a lens dislocated into the anterior chamber.

CORNEAL TRANSPLANTATION ON AN OPAQUE CORNEA. MR. TUDOR THOMAS.

In a man, aged 22, severe interstitial keratitis developed in each eye at 18 years of age. He afterward received a course of antisyphilitic treatment. In 1930 both corneas were opaque, and he could not count fingers held close to either eye. He could just discern hand movements, but could not see a person immediately in front of him.

The graft was taken from the cornea of an enucleated eye, which had received a perforating injury at the limbus twenty-six days previously, with retention of a nonmagnetic foreign body. A graft of the whole thickness of the cornea was then removed with trephine and scissors, its margins being cut in a shelving manner, and it was placed in sterile olive oil. The graft was just over 4 mm. in diameter.

The eye to be grafted was cocainized, atropine having been instilled beforehand. By means of a trephine somewhat larger than that used to remove the graft, a button was removed from the central portion of the cornea. Two cross-stitches were inserted into the corneal substance to hold the graft, which was then inserted. The eye was covered with a shield, and was treated daily with boric lotion and a mydriatic oil.

The graft united well and was nearly transparent ten days afterward. The eye was injected. Two days later the patient could count fingers at 1 meter. Examination with a loupe showed a central gray cloud in the deep layer of the graft, and slight encroachment on the margin by superficial blood vessels.

Three weeks after the operation the graft began to look dull, and, with the loupe, minute vessels could be seen in it, extending from the corneal stroma. Vision had deteriorated, and the patient could not count fingers. The graft became quite cloudy. Two months after the operation he was able to count fingers at 6 inches (15.2 cm.), and the opacity was mostly in the central area and in the deep layers, in which a few minute blood vessels could be seen. A year after the operation the graft was clearer than the surrounding cornea, though the latter was less opaque than before the operation. The patient could count fingers at a distance of 30 inches (76.2 cm.). His vision is now such that he can walk in London alone without using a stick.

DISCUSSION

MR. LESLIE PATON: The point which interested me chiefly in this connection is the possibility of grafts being taken from animals for use in human eyes. Many years ago I had such a case. The man had an extensive symblepharon resulting from a chemical burn, and the lower lid was adherent to the lower margin of the cornea. There was no lower fornix. I took the graft from the inside of a frog's mouth, with a successful result; the patient had a mobile eye, with a good lower conjunctival sac. My view is that the graft of mucous membrane formed a scaffolding on which healthy mucous membrane developed, and the raw surfaces, which were protected by the frog mucous membrane, were covered by conjunctiva which grew from the patient's own conjunctiva.

MISS IDA MANN: I also have grafted frog's mucous membrane on the human conjunctiva. On one occasion a man showed obliteration of the fornices following severe trachoma. The graft "took," and the patient had a good fornix for two months; then trachoma and shrinkage developed in the new conjunctiva.

MR. R. E. BICKERTON: One of the first grafts performed in this way was that done by Webster Fox, of Philadelphia, forty years ago. The graft adhered, but in three days became quite opaque; thereafter the patient had vision of 3/60.

Feb. 10, 1933

A. C. HUDSON, F.R.C.S., *President, in the Chair*

CONJUNCTIVITIS, PROBABLY TUBERCULOUS. MR. A. C. HUDSON.

In a woman, aged 29, the eye has the appearance of tubercle, but inoculation and other tests have given negative results for that disease. She apparently does not have a tuberculous lesion in any part of the body. In one eye the condition has been present for twenty-two months; in the fellow eye, for twenty months. Twenty-two months ago she injured one eye with chloride of lime. The lesions consisted of inflammatory thickening of the conjunctiva of the central part of the lower lids as far as the bulbar conjunctiva, associated with necrosis and a shallow ulceration. Then the upper lids became similarly affected. Healing was followed by much scar formation and by trichiasis. The left preauricular gland has remained enlarged and hard. The only pathogenic organism found was Staphylococcus aureus. No benefit followed the injection of a vaccine made from that organism.

DISCUSSION

MR. M. S. MAYOU: I think that the lesion is lupus, not ordinary tubercle.

CONGENITAL DISLOCATION OF THE LENS, WITH SUDDEN MARKED IMPROVEMENT OWING TO FURTHER DISLOCATION. MR. ADRIAN CADDY.

An unmarried woman, aged 29, had been troubled since childhood with a high degree of myopia and congenital dislocation of the lenses.

With a minus 18 diopter sphere and a minus 4 diopter cylinder before each eye, vision was 6/36. An operation was carried out, with little success. Last January, while in bed, she noticed that the vision of the right eye suddenly improved, and that the wearing of her spectacles spoiled her vision. The reason for this was that the lens had slipped into the vitreous. With a plus 4 sphere glass she had vision of 6/9. It was a case of natural couching of the lens. Nature had stepped in and done better than the surgeon.

DISCUSSION

MR. A. C. HUDSON: In cases in which the lens is couched, the character of the vitreous has an important bearing on the result. If the vitreous is fluid, the prospect of a successful result is greater than if the vitreous is normal.

CONGENITAL HYALINE MEMBRANES ON THE POSTERIOR SURFACE OF THE CORNEA. MISS IDA MANN.

The association of the formation of a hyaline membrane at the periphery of the posterior corneal surface with abnormality of the iris stroma and normal intra-ocular tension seems to be a rare congenital condition. In a man, aged 29, it involved the left eye only; visual acuity was normal, as was the tension, and the condition was seen accidentally. The membrane was applied to the posterior surface of the cornea around the greater part of the circumference. The anterior layer of the iris stroma was composed of abnormally thick strands which overlapped the margin of the pupil when this was dilated. The second case was that of a child aged 8 years, who showed a hyaline membrane on the deep surface of the cornea of both eyes and an abnormality of the iris stroma. Miss Mann had been unable to find a record of a similar case, although Sydney Stephenson, Coats and Herbert had discussed the question of the formation of hyaline material on the cornea. Ballantyne once showed a case which, in some respects, resembled that now being described. Miss Mann suggested that the condition was a purely developmental aberration arising from abnormality of the postendothelial tissue of the embryo.

DISCUSSION

MR. M. S. MAYOU: Several years ago, I showed a patient with congenital anterior synechiae and buphthalmos, in whom a band stretched across the anterior chamber. I now feel sure that this was an instance of a hyaline band. I agree with Miss Mann's explanation of her case.

SAFAR'S METHOD FOR TREATMENT OF DETACHMENT OF THE RETINA BY DIATHERMY. MR. J. COLE MARSHALL.

Safar aimed at getting discrete spots of coagulation in the choroidal layer in such a way that no injury was inflicted on the retina. There was no destruction or even weakening of the sclerotic. By reason of the needles employed and the small amount of current used, it seemed to be a more exact method than Larson's, as one avoided any serious damage beyond the point of puncture, especially in cases in which there were ruptures near the macula or near the ora serrata. By this method there was no need to resect muscles.

As soon as the diagnosis was certain, the patient wore Lochbrille spectacles, and he was kept in bed for the week preceding the operation. After operation the eyes were kept bandaged for fourteen days. The first dressing was carried out on the fourth day, and at the same time a quick examination of the fundus was made. It was important at this stage to prevent the patient from attempting to use the eye. The spectacles should still be worn and the patient should remain in bed until the end of the third week. The results might not appear until the end of the first week, and the retinal condition might continue to improve until the fourth week. A second intervention should not be essayed until at least a month had elapsed since the first operation.

Up to 1932, Safar had performed the operation in thirty-nine unselected cases, with reattachments in twenty-four.

HAND SLIT-LAMP. MR. N. BISHOP HARMAN.

This is an instrument of handy size, and its use seems simple in the hands of the inventor. The size and weight of the standard slit-lamp, he said, limits its use to the consulting room. The instrument submitted could be used as readily and easily as an electric ophthalmoscope, and with it an examination of children and of patients in bed could be made. It gave a valuable view of the media of the eye as deep as the anterior part of the vitreous, with a linear magnification of 12. A powerful condensing system produced an image of the filament on a slit fixed in the tube, and an image of the slit could be seen, free from any disturbing haze or irregularities of filament.

Book Reviews

Clinical Physiology of the Eye. By Francis Heed Adler, M.A., M.D., F.A.C.S., Instructor in Physiology and Ophthalmology, University of Pennsylvania School of Medicine; Assistant Surgeon, Wills Hospital. Price, $5. Pp. 405. New York: The Macmillan Company, 1933.

The American literature on ophthalmology has in the past been abundantly supplied with excellent general textbooks for medical students and specialists, but until recently it has been wofully deficient in authoritative treatises on the fundamental sciences which should be part of the elementary training of any one who intends to practice ophthalmology. Two classes of books are needed; textbooks for students, with which the student can acquire a general knowledge of the fundamental branches, and reference handbooks, through which the investigator and advanced student can find an orientation in relation to the current literature. The latter lie beyond the scope of any single author, and the need here is perhaps less urgent because adequate reference sources are available in foreign languages, but any one concerned in the training of young ophthalmologists must be familiar with the difficulties which an inadequate English literature places in the way of training in the fundamental preclinical branches of ophthalmology. It is not an accident, therefore, that in the past few years two such works have emanated from the faculty of the Post-Graduate School of Ophthalmology of the University of Pennsylvania, the first by Cowan, entitled "Elementary Ophthalmic Optics," and the second by Adler.

Dr. Adler has done a remarkably fine job in the preparation of this book. Admittedly, the field of the physiology of the eye is so large and so intimately connected with many branches of general physiology that any complete treatment of the subject would extend to work beyond the size of a useful and readable textbook for students. The author therefore states in his introduction that he has given more emphasis to those aspects in which he has been particularly interested. This decision requires no apology, since Dr. Adler has judiciously covered the broader realm in its essentials, and since his own investigations have made him a recognized authority in those branches of the subject that are of most intense current interest.

The book is divided into fourteen chapters. The first deals with the protective mechanism of the eye, including the blinking reflex, lacrimal mechanism, corneal and conjunctival sensation and permeability of the cornea. The second chapter describes the pupillary reactions and the mechanism of their control. Next follows a similar discussion of the mechanism of accommodation. The fourth chapter deals with the physical and chemical constitution of the lens and vitreous, together with those interesting problems of metabolism on which so much work has recently been done, the full fruition of which, it is hoped, will help one to understand the pathogenesis of cataract. The fifth chapter deals with the problem of visual acuity and the structural relations in the eye which

are required for its understanding. Next follows a discussion of the neuro-anatomy and neurophysiology of the visual mechanism from the retina to the cortex. The seventh chapter deals with the objective changes—chemical, mechanical and electrical—which occur in the retina on stimulation by light, while the eighth chapter deals with the corresponding subjective phenomena—dark adaptation, induction and color vision. Next comes a discussion of binocular vision and of entoptic phenomena. The eleventh chapter deals with the optic nerve, its action currents and metabolism. The succeeding chapter deals with the extra-ocular muscles, their coordination in the light of Sherrington's theory, nystagmus and the like. The two final chapters summarize the present knowledge regarding the formation and composition of the aqueous humor and the control of intra-ocular tension, and include a judicious comment on the contending theories in this field.

Throughout the book Dr. Adler draws on common clinical knowledge to illustrate and invigorate his discussion of purely physiologic matters, a procedure which makes the book especially useful to beginners in whom an interest in clinical ophthalmology is generally aroused before that in the fundamental sciences. Often this approach gives needed emphasis to some minor or less conspicuous clinical signs to which the clinician generally gives inadequate attention. This is particularly true in his discussion of the mechanism of pupillary control, which has been especially well presented.

The book is copiously documented with references to the literature which should serve as a guide for more advanced study. Necessarily, some difference of opinion must crop up between the author and the reader in the interpretation of the publications cited. Thus on page 28, Regnier is quoted as having found cocaine at a p_H of 8.7 seven times more powerful as an anesthetic than the usual acid solution. In my own experience cocaine hydrochloride was found to become insoluble at a p_H of 7.2; solutions of the degree of alkalinity mentioned by Regnier seem impossible.

On page 115 the author suggests, as the result of his own work and the calculations of Cowan, that diabetic changes in refraction may be due to a difference in the sugar content of the aqueous and vitreous. Undoubtedly the author has established the fact that the vitreous lags behind the aqueous in following changes in blood sugar levels. However, a computation based on Gullstrand's data for the normal eye shows that the myopia produced by an excess of dextrose in the aqueous over that in the vitreous of 1.2 per cent, which is more than twice the extreme range of variation in the severest diabetes, would amount to 0.05 diopter, while the hyperopia produced by the same excess of dextrose in the vitreous over that in the aqueous would be 0.16 diopter. A book must be well done, indeed, if on careful reading one can find no more important points of issue than these.

JONAS S. FRIEDENWALD.

Fuchs' Diseases of the Eye. Translated by E. V. L. Brown, Chicago. Tenth English edition. Price, $7. Pp. 612, with 255 illustrations and 41 colored plates. Philadelphia: J. B. Lippincott Company, 1933.

The appearance of a new edition of Fuchs' textbook has been awaited eagerly by American ophthalmologists. The last English edition was

published in 1924. Since that time both Professor Fuchs and the veteran translator, Alexander Duane, have died, but one feels that the demand for this great textbook will continue and will justify the publisher in undertaking this new translation. The choice of Dr. Brown, long a disciple of Professor Fuchs, translator of Salzmann's "Anatomy of the Eye" and an authority on the teaching of ophthalmology, to undertake this work was a fortunate one. The idea of the publisher and the translator was to produce a suitable and not too expensive textbook for American undergraduates. They have hence reduced the size of the present edition from 997 to 612 pages. The chapter on refraction written by Duane has been omitted, in accordance with the translator's belief that refraction should not be taught to American undergraduates. For the same reason the chapter on ophthalmic operations has been omitted. The chapters on anatomy, physiology, pathology and therapeutics have been omitted as special sections, but much of their material, especially that on anatomy, has been redistributed at the heads of various chapters. Thus 8 pages are devoted to the anatomy of the cornea, 14 to that of the uveal tract and 5 to that of the retina and the optic nerve in the corresponding chapters, an arrangement which has obvious advantages. In this arrangement and in all other respects the author has closely followed the fifteenth German edition as revised by Professor Salzmann. The colored pictures of the fundus are an important addition to this edition. Forty-one of the pictures, drawn by Salzmann and very well reproduced, illustrate most of the common and some of the less common ophthalmoscopic conditions. They replace the twelve plates of the fundus in previous editions. A valuable chapter on the central visual pathways has been added to this edition, apparently Professor Salzmann's contribution.

Duane's well known chapter on motor anomalies, which was a feature of later English editions, has been replaced by one half as long, but it is excellent in its clearness and choice of the most important matter.

It is hardly necessary to emphasize the qualities which have made Fuchs' textbook a classic in the teaching of ophthalmology all over the world. Fuchs was essentially a great clinician, and his valuable discussions of pathology are not allowed to crowd out his clear and vivid descriptions of the clinical findings necessary for diagnosis, descriptions which have never been excelled.

This translation is more literal than that of Duane, occasional instances of quite Germanic structure being carried over into the English text. It is apparently a completely new translation and, on the whole, is very clear and readable.

One might criticize the absence of translator's notes which would bring certain subjects up to date. Thus it is strange to see the old description of rheumatic iritis given without any mention of focal infection by one who knows focal infection as Dr. Brown does, while in the Duane edition so-called rheumatic iritis and gonococcal iritis were grouped as focal iritis with proper reference to the importance of various common foci. Another classic concept which seems to require a note of modification is Fuchs' belief that closure of the central retinal artery is nearly always due to an embolus. The work of Harms and

Scheerer seems to leave no room for doubt that many if not most of such cases are due to endarteritis and other causes. One might also object to Fuchs' statement that "Therapy is as good as powerless against Sympathetic Ophthalmia, once it has broken out."

On the whole, however, one need hardly quarrel with an authority who was right as often as was Professor Fuchs. Dr. Brown, in following the word of the master as something almost sacred, has rendered English-speaking ophthalmologists a signal service.

S. R. GIFFORD.

The Opticians' Dispensing Manual. By Edward T. Hartinger. Price, $3.50. Pp. 192, with 92 figures. New York: Edward T. Hartinger, 1932.

While this book is intended primarily for those who are concerned with the grinding of lenses and the fitting and sale of various types of lenses and frames, it contains much information with which the ophthalmologist should have at least a cursory acquaintance. Moreover, as the author states in his preface, his theories can be applied with merely the aid of a pencil, cardboard, a caliper and a ruler.

The first chapters deal with Oxfords, spectacles and the adjusting of frames. While Mr. Hartinger seems overly fond of Oxfords, he is not unnecessarily prejudiced in their favor.

The chapters on lenses, transpositions and bifocals are of especial value, particularly those in which he mentions some impossible prescriptions that are received, such as decentering a cylinder with a horizontal axis to obtain the effect of a prism base in or base out.

His chapter on transposition of compound lenses could have been simplified. If a = sphere (+ or —) and b = cylinder (+ or —) in the original prescription, then, in the transposition, the new sphere =a + b (algebraic sum), and the new cylinder = —b (algebraically) to the opposite axis. For crossed cylinders, a = o, and if both cylinders are of the same sign, both cylinders are converted to a compound lens, while if they are of opposite sign, only one cylinder is to be so converted before adding together the new value or values of the combination.

The book as a whole deserves commendation. Mr. Hartinger seems to be thoroughly conversant with his subject.

W. F. DUGGAN.

Bulletin of the Ophthalmological Society of Egypt. Volume XXV. Session 29. Cairo, Egypt: Misr Press, 1932.

This volume contains articles on subjects which are striking but not uncommon. Tattooing of the cornea with platinum chloride, a comparison of the Gonin and Sourdille operations for retinal detachment, vernal catarrh, trachoma, atrophy of the iris in glaucoma, thrombosis of the central vein of the retina in a 21 year old girl (a tuberculin test seems to have been indicated in this case), pulsating exophthalmos and an analysis of pituitary tumors are included in the communications of the society.

Prof. Sayed A. H. Bey and Dr. M. A. H. Attiah report that Jacobson's solution (a 3.6 per cent solution of benzyl-cinnamate in oil), when given intramuscularly, seems to have no effect on trachoma.

Dr. A. F. Tobgy also reports that copper salts, when given intravenously, do not influence the healing of trachomatous lesions.

There is an article by Dr. Rowland Wilson ("A Short Slitlamp Study on the Corneal Vessels in Egyptian Trachoma") which has already appeared in the *Folia ophthalmologica orientalis*.

W. F. DUGGAN.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Prof. F. de Lapersonne, 217, Faubourg St. Honoré, Paris.
Secretary-General pro tem.: Dr. A. Churchill, 66, Boulevard St. Michel, Paris.

International Ophthalmologic Congress

Secretary: Dr. Francisco Poyales, Olozaga 3, Madrid.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. Frank H. Crawley, 5 Fitzwilliam Pl., Dublin.
Place: Dublin. Time: July, 1933.

Ophthalmological Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgoam Road, Bombay 4.
Secretary: Dr. C. R. Athawale, Bhatwadi Girgoam, Bombay 4.
Place: A. M. C. Free Ophthalmic Hospital Bldg., Poi Bawadi Parel, Bombay 12.
Time: First Friday of every month.

Ophthalmological Society of the United Kingdom

President: Mr. A. H. H. Sinclair, 6 Charlotte Square, Edinburgh.
Secretary: Mr. Humphrey Neame, 56 Portland Place, London, W. I.

Oxford Ophthalmological Congress

Master: Dr. Cyril Walker, Bristol, England.
Secretary: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Place: Oxford, England. Time: July 5-8, 1933.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. A. C. Hudson, 50, Queen Anne St., W. 1, London.
Secretary: Dr. J. D. Magor Cardell, 27, Weymouth St., W. 1, London.
Place: London. Time: June 9, 1933.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.
Place: Paris. Time: June 26, 1933.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.
Place: Heidelberg. Time: Aug. 3-5, 1933.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. Frederick H. Verhoeff, 82 Commonwealth Ave., Boston.
Secretary: Dr. Parker Heath, 1551 Woodward Ave., Detroit.
Place: Milwaukee. Time: June 12-16, 1933.

*** Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.**

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
President-Elect: Dr. J. M. Wheeler, 30 W. 59th St., New York.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Boston. Time: Sept. 18-22, 1933.

American Ophthalmological Society

President: Dr. Thomas B. Holloway, 1819 Chestnut St., Philadelphia.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.

SECTIONAL

New England Ophthalmological Society

President: Dr. Hugo B. C. Riemer, 128 Newbury St., Boston.
Secretary-Treasurer: Dr. Benjamin Sachs, 483 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. Hans Barkan, 490 Post St., San Francisco.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: San Francisco. Time: June 28-30, 1933.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. R. Secoy, 1802 Hewitt Ave., Everett, Wash.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: A. R. McKinney, 703 Second National Bank Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. John B. Gregg, 101 S. Main St., Sioux Falls, S. Dakota.
Secretary-Treasurer: Dr. F. H. Roost, 420 Trimble Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Secretary: Dr. William A. Wagner, 200 Carondelet St., New Orleans.
Place: Richmond, Va. Time: November, 1933.

Southwestern Michigan Triological Society

President: Dr. Carl Snapp, 604 Medical Arts Bldg., Grand Rapids.
Secretary-Treasurer: Dr. Lee O. Grant, 420 Medical Arts Bldg., Grand Rapids.
Time: Third Thursday of alternate months.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Ralph W. Danielson, 258 Metropolitan Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. J. J. Cohane, 59 College St., New Haven.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Thomas H. Hall, Jr., 617 Mulberry St., Macon.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. Robert J. Masters, 805 Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: December 13, 1933.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. W. Pearson, 410 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. W. F. Boiler, 105 E. Iowa Ave., Iowa City.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Ray Connor, 28 W. Adams Ave., Detroit.
Secretary: Dr. Ralph B. Fast, 136 E. Michigan Ave., Kalamazoo.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Harold J. Rothschild, 239 Lowry Bldg., St. Paul.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Lawrence G. Dunlap, 101 Main St., Anaconda.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.
Place: Anaconda. Time: July 11, 1933 (meetings are held semi-annually).

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Charles H. Schlichter, 556 N. Broad St., Elizabeth.
Secretary: Dr. B. E. Failing, 31 Lincoln Park, Newark.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.
Secretary: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Axel Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

President: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Walter Bristow, Medical Bldg., Columbia.
Secretary: Dr. J. W. Jervey, Jr., 101 Church St., Greenville.
Place: Columbia. Time: October, 1933.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. D. Blassingame, 899 Madison Ave., Memphis.
Secretary-Treasurer: Dr. Ralph O. Rychener, 130 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. O. S. Hodges, 388 Pearl St., Beaumont.
Secretary: Dr. A. F. Clark, 1034 Nix Professional Bldg., San Antonio.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. T. Hasler, 290 W. Center St., Provo.
Secretary-Treasurer: Dr. T. F. Welsh, 920 Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Frank P. Swart, 142 W. York St., Norfolk.
Secretary-Treasurer: Dr. H. G. Preston, 307 Professional Bldg., Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.
Secretary: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Elbert A. Curtis, 65 Central St., Newark.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ACADEMY OF MEDICINE OF TORONTO, SECTION OF OPHTHALMOLOGY

Chairman: Dr. P. J. F. Houston, 34 Hurndale Ave., Toronto.
Secretary: Dr. Alexander E. MacDonald, 151 Bloor St., W., Toronto.
Time: First Monday of winter months.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. G. E. Clay, 384 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. William M. Rowland, 906 Cathedral St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. W. F. C. Steinbugler, 515 Ocean Ave., Brooklyn.
Secretary: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. H. D. Andrews, 33 Allen St., Buffalo.
Secretary-Treasurer: Dr. Thurber Le-Win, 112 Linwood Ave., Buffalo.

Chicago Ophthalmological Society

President: Dr. Dwight C. Orcutt, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medical and Dental Arts Club. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. Justin M. Waugh, Cleveland Clinic, Cleveland.
Secretary: Dr. James T. Collins, 1105 Medical Arts Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. Roy B. Metz, Hanna Bldg., Cleveland.
Secretary: Dr. Paul G. Moore, Medical Arts Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. L. F. Appleman, 308 S. Sixteenth St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. John B. Alcorn, 167 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Edward W. Harris, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. O. M. Marchman, 1717 Pacific Ave., Dallas, Texas.
Recording Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Corresponding Secretary: Dr. J. M. Potts, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. Cecil C. Jones, 1121 Equitable Bldg., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. R. H. Seeley, 9 Court St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Valin R. Woodward, Flatiron Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7.30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, 26 Sheldon Ave., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 320 Medical Arts Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. J. F. Gamble, 201 Main St., Houston, Texas.
Secretary: Dr. B. F. Payne, 1304 Walker Ave., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Albert N. Lemoine, 906 Grand Ave. ,Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Ferris L. Arnold, 923 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Clyde E. Harner, 312 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from September to June.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. D. E. Godwin, 910 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Elks Temple. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. Karl N. Victor, Breslin Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Place: French Village. Time: Second Thursday of each month, September to May, inclusive, at 6:30 p. m.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. Albert P. Tibbets, 1801 I St., Washington.
Secretary: Dr. G. Victor Simpson, 1710 Rhode Island Ave., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Samuel G. Higgins, 324 E. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Montreal Ophthalmological Society

President: Dr. J. P. E. Bousquet, 66 Sherbrooke St., West, Montreal, Canada.
Secretary: Dr. J. A. MacMillan, 1410 Stanley St., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Eugene Orr, 706 Church St., Nashville, Tenn.
Secretary-Treasurer: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Secretary-Treasurer: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Algernon B. Reese, 73 E. Seventy-First St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. L. G. Howard, 532 First Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. R. W. Vaughan, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. W. E. Munroe, 277 Alexander St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Max W. Jacobs, Missouri Theatre Bldg., St. Louis.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Robert E. Parrish, 712 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. King Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. George N. Hosford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. T. D. Boaz, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. A. Veasey, Jr., 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. J. L. Walter, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. G. Dickinson, 504 State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. Harold H. Joy, 504 State Tower Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy Dunlap, Medical Arts Bldg., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, Medical Arts Bldg., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.

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